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THE EARLY DIAGNOSIS OF RHEUMATIC HEART DISEASE IN CHILDREN*

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In spite of the extensive literature on rheumatic heart disease, there is still much to be learned regarding the natural history and epidemiology of rheumatism in juvenile patients and the methods of recognizing valvular disease in the earliest stages.

Our purpose in this paper is threefold: (1) to classify all patients admitted between the years 1923 and 1930 to the Minneapolis Lymanhurst Clinic for children; (2) to offer a scheme for the differential diagnosis of heart disease, and (3) to discuss and relate in the order of their importance the five chief methods of diagnosis, namely, (*a*) etiologic, (*b*) structural or anatomic, (*c*) functional, (*d*) roentgenologic and (*e*) electrocardiographic.

During the period from 1923 to 1930, 809 children between the ages of 5 and 16 were admitted to the Minneapolis Lymanhurst Heart Clinic. These children were referred to the clinic because of signs and symptoms indicative of heart disease or because of so-called rheumatic manifestations.

The patients with conditions diagnosed as "organic heart disease" were observed for at least one year, and ninety-four of them have remained under observation for three years or more. In all cases at least two roentgen examinations were made. Other methods of diagnosis, such as the esophagram, the electrocardiogram and functional tests were used whenever necessary. Patients with conditions diagnosed as "no heart disease" were also observed over a period of at least three months. In these cases, too, one or more roentgenograms were obtained. The diagnosis of organic heart disease was agreed to by two of us (Seham and Shapiro).

CLASSIFICATION OF CASES IN GROUP WITH ORGANIC HEART DISEASE

Table 1 shows that less than one half of the children admitted actually suffered from organic heart disease. The incidence of organic

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heart disease was 46 per cent (table 1*A*) against 54 per cent (table 1*B*) of the patients in whom no organic lesion could be ascertained. The diagnoses listed in this table represent the final diagnoses and not the ones made on first contact.

TABLE 1.—*Patients with and Those without Heart Disease*

	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent
A. Organic heart disease					379	46
1. Congenital			69	18.0		
(a) Congenital lesions (patency predominating)	59	85.0				
(b) Congenital lesions (stenosis predominating)	8	11.0				
(c) Transposition of greater vessels	1	2.0				
(d) Dextrocardia	1	2.0				
Total	69	100.0				
2. Rheumatic			281	74.0		
(a) Mitral regurgitation (rheumatic and systolic murmur	127	45.0				
(b) Double mitral disease (mitral regurgitation and stenosis)	114	40.0				
(c) Mitral disease and aortic regurgitation	30	9.0				
(d) Aortic regurgitation	5	3.0				
(e) Mitral stenosis	1	0.35				
(f) Pericarditis	4	2.65				
Total	281	100.0				
3. Miscellaneous			12	3.6		
(a) Essential hypertension	2	16.0				
(b) Toxic goiter (hyperthyroidism)	2	16.0				
(c) Diphtheritic myocarditis	2	16.0				
(d) Hypertrophy of heart (idiopathic)	1	9.0				
(e) Subacute bacterial endocarditis	5	43.0				
Total	12	100.0				
4. Undiagnosed			17	4.4		
Total			379	100.0		
B. No heart disease					430	54
1. Rheumatism without carditis			226	53.0		
(a) Rheumatic fever (b) Chorea (c) Scarlet fever	45	19.0				
(d) Rheumatism; symptoms of heart disease	19	8.5				
(e) Rheumatism and systolic murmur	35	15.5				
(f) Growing pains alone	43	19.5				
(g) Growing pains, systolic murmur, symptoms of cardiac disease	9	4.0				
(h) Growing pains, signs and symptoms of cardiac disease	64	28.5				
(i) Growing pains and systolic murmur	11	5.0				
Total	226	100.0				
2. Signs and symptoms of heart disease; no rheumatism			76	17.0		
(a) Fatigue (b) Dyspnea (c) Loss of weight and strength (d) Tachycardia (e) Palpitation (f) Precordial pain (g) Radiation of pain (h) Nausea and vomiting (i) Gastric and abdominal pain (j) Cough (k) Edema (l) Giddiness (m) Headache (n) Flushing (o) Nosebleed (p) Syncope						
3. Systolic murmur alone			68	16.6		
4. Systolic murmur, circulatory signs and symptoms			27	6.0		
5. Functional arrhythmias			27	6.0		
6. Acrocyanosis			4	0.9		
7. Bronchial asthma			2	0.5		
Total			430	100.0		

Congenital Lesions.—Of the 379 children with organic heart disease, 18 per cent revealed a congenital lesion (table 1*A*, 1). A definite, constant thrill was felt in 41 per cent, and the roentgen shadows were abnormal in 70 per cent. The electrocardiogram assisted in the clinical diagnosis in many instances.

It is true that classifications of heart disease in the adult do not show such a high incidence of congenital lesions. The most severe forms are rather rare in the adult, since the majority of persons with these severe forms do not live beyond puberty. Moreover, with increasing age the history becomes less reliable, and changes occur in the heart that make the clinical picture less typical. Therefore, it becomes more difficult to recognize the disease later in life.

Rheumatism.—Whereas in a clinic for adults with heart disease, rheumatism is said to be the cause of about from 30 to 35 per cent of all cases, we found it to be responsible for 74 per cent of our cases (table 1*A*, 2). This increased number may be due to the fact that a positive history is more likely to be obtained shortly after an attack and also because rheumatism occurs chiefly before the age of puberty.

Of the rheumatic children, 45 per cent suffered from mitral regurgitation (table 1*A*, 2*a*). Of these, 92 per cent gave a history of rheumatism (table 2, 3). All of them showed systolic murmurs at the apex; in 42 per cent abnormal roentgen shadows were seen on the first contact, and in 70 per cent, by the end of the third year. Esophagrams were taken in only twenty-two cases; of these, 55 per cent showed abnormal films.

Double mitral disease was present in 40 per cent of the patients (table 1*A*, 2*b*). In these cases the diagnosis was made because of the history of rheumatism and a double murmur (mid-diastolic, presystolic or both), and in a few cases because of a presystolic thrill. Along with the presystolic murmur was the usual rumble, ending with a snap at the apex or at the left of the sternum. Over the pulmonary area the second sound was usually accentuated and often reduplicated. In the majority of the records we found roentgenologic evidence of abnormality, which included posterior compression and, in a few instances, dislocation of the esophagus to the right. In 9 per cent of the cases, a diagnosis of double mitral disease and aortic regurgitation was made (table 1*A*, 2*c*). Only 3 per cent were found to have aortic disease alone (table 1*A*, 2*d*). They were characterized by the high pitched diastolic murmur heard best along the left border of the sternum in the third and fourth interspaces, by peripheral signs and by a left ventricular type of roentgen shadow. There was only one case of pure mitral stenosis (table 1*A*, 2*e*). The four cases of pericarditis (table 1*A*, 2*f*) were not primary, but are classified as such because the signs and symptoms of pericarditis were predominant.

Miscellaneous and Undiagnosed Cases (Table 1A, 3 and 4).—Whereas in the adult essential hypertension is responsible for more than 50 per cent of all cases of organic heart disease, in the child it is rather an insignificant cause. In our series only two of the total number of cases were diagnosed as hypertension. The same is true of all other forms of nonrheumatic heart disease. Of the five cases of subacute bacterial endocarditis, three were originally rheumatic. Therefore, we did not feel justified in listing them under rheumatism: 4.4 per cent of the total are classed as undiagnosed (table 1*A*, 4). We are reasonably certain that these cases were organic in origin. However, we were unable to decide whether they were congenital, acquired or both, and we could not make a definite structural diagnosis.

CLASSIFICATION OF CASES IN GROUP WITH NO HEART DISEASE

Another important difference between heart disease in the adult and that in the child lies in the large number of so-called functional signs and symptoms of the disease that children suffer from that are not due to organic causes. The diagnosis for 54 per cent of all the children admitted was "no heart disease" (table 1*B*). No attempt was made toward an etiologic classification. Most of the disorders listed are merely the complaints of the children themselves or the objective signs noted by the school nurse and the physician.

Rheumatism Without Carditis.—Fifty-three per cent of these conditions are cases of "potential" heart disease in which the diagnosis of heart disease could not be made. The patients suffered from rheumatism, but they did not have carditis. In this group are included patients with all forms of rheumatism, with or without a systolic murmur and with or without signs and symptoms simulating heart disease. In 19 per cent of the cases the patients showed no symptoms or signs other than a history of having had one of the rheumatic triad (table 1*B*, *a* to *c*).

Signs and Symptoms.—In group 2 of table 1*B* are listed the children who gave no history of rheumatism, but who complained of one or more of the functional disorders (mentioned under the subdivisions from *a* to *p*) or who were excused from activities in the gymnasium on the grounds of signs and symptoms indicative of heart disease. Groups 3 and 4 in table 1*B* account for almost 23 per cent of the cases in which the diagnosis was "no heart disease." Some of these patients had systolic murmurs alone that proved to be nonpathologic, and others revealed signs and symptoms as well as a systolic murmur. Group 5 comprises cases of sinus arrhythmia, extrasystole and a case of paroxysmal tachycardia. It is hardly necessary to discuss groups 6 and 7.

While the differential diagnosis between the later stages of organic heart disease and no heart disease is relatively simple, there remains a need for a procedure that would render more easy and accurate the

differential diagnosis between what we have labeled "early rheumatic heart disease" and the other forms of functional symptom complexes that simulate heart disease.

Because it is difficult to separate the early from the later stages by clinical methods, it is necessary to admit that the use of the term "early" is arbitrary. What we really mean in this article by "early" is "early as possible" or "earliest." We realize that from the standpoint of damage done to the heart and the symptoms produced, aortic regurgitation or mitral stenosis alone may not represent a later stage than

TABLE 2.—*The Differential Diagnosis of: (1) No Heart Disease; (2) Murmur and Rheumatism (Unproved); (3) Early Mitral Disease; (4) Congenital Heart Disease (Patencies)*

	Diagnosis			
Means of Diagnosis	1 No Heart Disease (89) per Cent	2 Murmur and Rheumatism (Unproved) (24) per Cent	3 Early Mitral Disease (94) per Cent	4 Congenital Heart Disease (Patencies) (53) per Cent
1. Rheumatism	0	100	92	2
2. Growing pains	50	35	55	43
3. Fever	6	4	4	0
4. Tachycardia	4	8	4	1
5. Thrill	0	0	0	41
6. Pulmonary congestion	0	0	2	5
7. Circulatory signs	37	33	45	38
8. Weight-height index, 10 per cent below	45	50	38	45
9. Murmur	Not diagnostic	Not diagnostic	Not diagnostic	More diagnostic
10. Abnormal roentgen observations	4	0	42	70
11. Abnormal esophographic observations	9	0	55 (22 cases)	5
12. Abnormal electrocardiograms	0	0	Myocardial changes; absent iso-electric period	High excursions; right ventricular preponderance

a simple mitral regurgitation, but because in our series aortic regurgitation alone occurred in only 3 per cent, and because mitral stenosis alone was present in only 0.35 per cent, we have felt justified in referring to mitral regurgitation alone or to the combination of a rheumatic history and systolic murmur that later proved to be organic heart disease as the "early stages." In other words, we think that the clinical problem is stated as accurately as possible when we say that a diagnosis of mitral regurgitation with or without roentgen evidence of enlargement and without decompensation best represents the early or earliest stages of rheumatic heart disease.

As an attempt in this direction, we offer the classification presented in table 2. On the left are recorded the various means of diagnosis. In the first column are listed eighty-nine cases in which there was no history of rheumatism, but in which the patients on admission to our

clinic presented a systolic murmur at the apex or at the base of the heart, with or without other signs and symptoms, which after at least one year of close study proved to be nonpathologic. In the next column are listed the cases of twenty-four children who were admitted as "potentials"; that is, they had had rheumatism and the usual systolic murmur; some showed symptoms and some did not, but none of them could be proved to have organic heart disease after a year's observation. These patients were true "potentials" who at least over a period of one year had escaped endocarditis. In the next group there were ninety-four cases diagnosed as mitral regurgitation or early mitral disease. All of these were studied for three years or more. In some a final diagnosis could not be made until later; in the majority the final diagnosis confirmed that of the first contact (within one month after admission). The data presented in this column represent the analysis of the records of the first contact. The last column represents the group of children with congenital heart disease. We did not include here "blue children" or those in whom there was a predominance of signs and symptoms of stenosis.

Rheumatism.—Our definition of rheumatism is limited to rheumatic fever, chorea and scarlet fever complicated by rheumatic fever. Obviously, the cases in the first column (table 2) were selected because there was no history of rheumatism. The second and third columns differ from one another only in the progress of the condition. The former remain "potential," and the latter remain organic heart disease. It is possible that some of the second group will ultimately change into group 3. As can be seen, a history of rheumatism is of great importance in differentiating between early mitral disease and congenital heart disease.

Growing Pains.—We purposely separate growing pains from all other forms of rheumatism in this table. We consider as growing pains those vague and general discomforts usually unassociated with fever, occurring chiefly in the legs and arms and sometimes in the joints, the abdomen or other muscles of the body. We have grouped them all under the heading of "growing pains." This is an arbitrary definition and needs finer clinical differentiation. As found in table 2, a history of growing pains is of no value in showing whether heart disease is present or not. Growing pains were practically as frequent in children who did not have organic heart disease as in those in whom a diagnosis of rheumatic heart disease was made. A comparison of the incidence of growing pains as shown in columns 1 and 2 seems to indicate that growing pains occur frequently in children, and that they are as frequently nonrheumatic as they are rheumatic. (A study of growing pains is being made at the present time.)

Fever.—Not infrequently one reads that fever is an important index of active rheumatism, and it is suggested by many writers, especially the English, that fever is an important sign of early rheumatic endocarditis. We have not found that fever is a frequent sign in the early stages of the disease. It is true that this sign is rather persistent in many children in whom a definite diagnosis of organic heart disease has already been made, as for example in children with double mitral disease or in those who have had an attack of decompensation. In the ninety-four cases of early mitral disease (group 3) we took the temperatures by mouth (the rectal temperature is more accurate) repeatedly over long periods; we found only four that showed a continuous and recurrent fever of over 100 F. This incidence is no higher than that in children who did not have heart disease. However, this question needs more thorough and continuous study.

Tachycardia.—Tachycardia is also frequently considered an important sign of active rheumatism and therefore useful in the diagnosis of rheumatic endocarditis. The pulse rate is so sensitive and so subject to external influences that it is difficult to be certain as to when tachycardia is significant. Our routine method was to take the pulse after the child had become accustomed to his environment. The pulse readings of the first two visits were excluded. A pulse rate of over 100 in a child 6 years of age or older was considered abnormally fast. Our data on this point show practically no difference in the incidence of tachycardia between the cases included in the first column and those included in the third column.

Thrill.—A thrill definitely felt at repeated intervals is usually considered to be evidence of the presence of organic disease. It seems to be of great value in differentiating congenital heart disease from the other three groups. It is especially important in the differentiation of congenital heart disease from early mitral disease. In 41 per cent of the cases of congenital lesions of the heart we were able to feel a systolic thrill, usually at the base and sometimes at the apex.

Pulmonary Congestion.—In a small percentage of patients with acquired and congenital heart disease, signs of pulmonary stasis were found by auscultation, by roentgen examination or by both.

Circulatory Signs.—In the adult circulatory signs and symptoms are more significant of organic changes than in the child. As can be seen from our data, the presence of such circulatory signs as are enumerated in table 1 *B* (*a* to *p*) are of little value in the diagnosis of early heart disease. Many children who do not have heart disease are malnourished, nervous and fatigued and complain of many and various common functional disorders that simulate heart disease. In

all four groups the incidence of these disorders is about the same. A difference of 8 per cent is not of sufficient importance to be used for diagnosis.

The Weight-Height Index.—We used 10 per cent below the average weight-height index (Woodbury-Holt) as the index of underweight. The same thing holds true of the weight-height index as of the circulatory signs. Of the group that did not have heart disease 45 per cent were 10 per cent underweight, as compared to 38 per cent of the group with organic heart disease. It is true that in the later stages of heart disease in those included under the heading "all others" the state of nutrition was usually poor, but in these early cases the majority of the children were in a good physical condition. In the early stages the progress may be slow, and while the children are free from active infection the muscles of the heart adjust themselves adequately for a long period without allowing abnormal changes in the metabolism of the body.

Murmur.—The rôle of the murmur in diagnosis is rather difficult to evaluate. Of course it is impossible to make a statistical study of the presence of a murmur, the kind of murmur or its quality and its transmission, because our means of interpretation of the characteristics of murmurs are so subjective. The only way in which the subjective element can be eliminated is by the use of the phonocardiograph.

A great deal has been written on the differentiation between the functional systolic murmur and the organic murmur. The textbooks on heart disease attempt to differentiate between the two on the basis of the length of the murmur, the point of maximum intensity and the transmission. It is true that in some instances in the early stages of rheumatic heart disease there is a characteristic long blowing holosystolic murmur transmitted into the axilla, the point of maximum intensity being at the apex, but there are so many exceptions to this rule that we are reluctant to consider the murmur of great diagnostic significance. This is especially true in the very early stages during or within a month or two after the first attack of rheumatism. During this period the murmur is inconstant; the records show that at times the murmur is heard and at other times it is absent. At this time it is frequently merely a prolongation of the first sound, and sometimes the heart sounds seem to be fainter than they should be. If children with these conditions are examined repeatedly over a long period, definite evidence of organic changes accumulates.

In spite of the fact that a great deal has been written about the different features of organic and functional systolic murmurs, and

even though the character of the former may in some instances be highly indicative, we feel justified from the observations that we have made in this group of cases in the conclusion that both in their character and in their propagation organic murmurs may resemble functional murmurs so closely that it is not possible to differentiate them in all cases.

There is another aspect of this question. Many writers, especially the English, are prone to suspect rheumatism as the chief cause of systolic murmurs in debilitated children. We found, however, as is shown in sections 7 and 8 in table 2, that asthenic, malnourished children and those otherwise below par made up the majority of those who were suspected of having heart disease but who did not have it. We believe that the murmur is of more diagnostic importance in congenital heart disease. There is something bizarre and peculiar about these murmurs that places them apart from other systolic murmurs.

Roentgen Diagnosis.—We cannot understand how writers can state that the x-ray is of little value in the diagnosis of rheumatic heart disease. It is true that it is not of such differential value when it is necessary to decide whether the patient does not have heart disease or whether the condition is in its later stages, but we have found that it is of special value in the diagnosis of the early stages. This is obvious in table 2. Whereas in the first two columns abnormalities revealed by the x-rays were practically never found (the 4 per cent in the first column falls within normal error), in the third column 48 per cent of the cases were diagnosed by the roentgenologist as abnormal. It is true that his diagnosis as to the specific type of lesion was frequently wrong, but in almost one half of the patients, the roentgen diagnosis confirmed the other usual clinical methods. An even higher percentage of cases (70 per cent) were diagnosed as abnormal by the roentgenologists in the group of patients with congenital heart disease.

Before attempting to estimate the value of a roentgenologic diagnosis in relation to the thoracic index and of these two factors in relation to the clinical diagnosis, it is first necessary to consider what is a normal cardiothoracic index. It has been customary on the part of practically all roentgenologists to consider a cardiothoracic index of over 50 per cent in the adult as an indication of an enlarged heart. This is supposed to represent the relation of the transverse diameter of the heart at the widest level, which is usually that of the right ventricles, to the widest diameter of the chest. The left auricles, the median convexity and the conus pulmonaris are not included in the measurement. In order to determine whether the same index can be used for children in this study, we compared the data on two groups

of normal children, those of Kirsch [1] and of Lincoln.[2] The former studied this question by the orthodiagraphic method, and the latter used roentgenograms taken at a distance of 6 feet (182.9 cm.). In table 3 the means and the standard deviations of the two authors

TABLE 3.—*The Relation Between the Transverse Diameter of the Heart and That of the Chest in Normal Children*

		Lincoln-Spillman			Kirsch			Combined		
Age	Sex	No.	Mean	Standard Deviation	No.	Mean	Standard Deviation	No.	Mean	Standard Deviation
2	M	7	49	2.0	2	50.77	..			
	F	5	45	2.0	..	.....	..	9	49.39	..
3	M	11	47	3.5	7	50.26	..	14	47.82	..
	F	7	47	3.2	5	49.79	..			
4	M	23	48	2.5	3	49.72	..	18	48.27	..
	F	24	49	2.5	2	51.98	..	12	48.15	..
								30	48.22	..
5	M	38	48	3.4	4	49.85	..	26	48.27	..
	F	36	48	2.7	2	49.37	..	26	41.23	..
								52	48.71	..
6	M	35	47	3.9	3	46.54	..	42	48.17	..
	F	32	48	3.3	8	49.94	..	38	48.07	..
								80	48.12	..
7	M	35	46	3.0	6	49.36	..	38	46.96	..
	F	35	47	3.1	7	51.33	..	40	48.39	..
								78	47.69	..
8	M	31	46	3.5	8	47.22	..	41	46.49	..
	F	33	47	3.2	2	53.21	..	42	47.72	..
								83	47.11	..
9	M	33	45	3.6	4	49.23	..	39	46.35	..
	F	27	46	2.7	1	47.81	..	35	47.35	..
								74	46.82	..
10	M	21	46	3.3	7	49.73	..	37	45.46	..
	F	19	46	2.9	2	46.95	..	28	46.06	..
								65	45.72	..
11	M	19	46	2.7	8	49.15	..	28	46.93	..
	F	23	45	4.3	4	49.41	..	21	46.09	..
								49	46.57	..
12	M	15	43	2.7	10	50.92	..	27	46.93	..
	F	20	45	4.3	17	48.97	..	27	45.65	..
								54	46.39	..
13	M	9	52	8.0	9	51.40	..	25	46.17	..
	F	8	46	4.0	8	49.74	..	37	46.82	..
								62	46.56	..
14	M	..	..	...	5	48.32	..	18	51.70	..
	F	..	..	...	3	49.81	..	16	47.87	..
								34	49.90	..
15	M	..	..	...	2	48.54	..			
16	M	..	..	...	1	44.16	..			
17	M	..	..	...	1	51.12	..			
	F	..	..	...	1	50.94	..			
Total		534			127			661		
Average			47	3.34		49.72	2.62		47.32	3.76

are compared. Using their data, we calculated the combined means and the standard deviations. As can be seen in table 3, there is a

1. Kirsch, Oscar: Grundlagen der orthodiagraphischem Herz grössen- und Thoraxbreitenbeurteilung im Kindesalter, Berlin, S. Karger, 1929.

2. Lincoln, E. M., and Spillman, R.: Studies on the Hearts of Normal Children: II. Roentgen Ray Studies, Am. J. Dis. Child. **35**:791 (May) 1928.

remarkable similarity between the two. The mean that we obtained from Kirsch's data for all children of all ages is 40.72 per cent, with a standard deviation of ± 2.6; that of Lincoln is 47 per cent, with a standard deviation of ± 3.34. Although Kirsch's work is theoretically more accurate, the orthodiagraph having been used, the small number of children examined makes the accuracy of his standard deviation questionable. However, in spite of the difference in method and of the numbers studied there is a reliable consistency between the two sets of figures. The combined means of the two studies representing

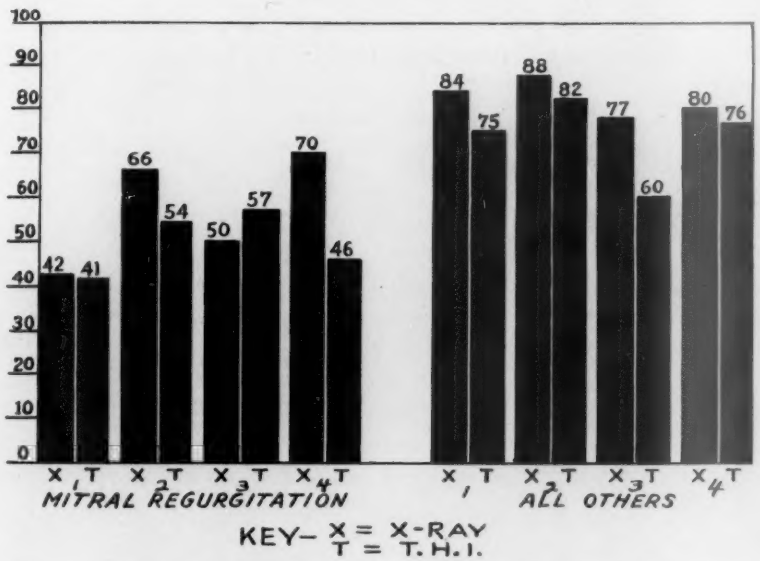

Fig. 1.—The percentage relation between abnormalities revealed by the x-ray and the cardiothoracic index in heart disease. *x* represents the x-ray picture; *t* the cardiothoracic index.

661 children is 47.2 per cent for boys, 47.455 per cent for girls and 47.32 per cent for both. The standard deviation for the boys (it was not thought necessary to calculate that of the girls because they were so nearly alike) was ± 3.76 per cent. In terms that can be applied to diagnosis, this means that 66 per cent of all normal children have a cardiothoracic index between about 44 and 50 per cent, and in the remaining 33 per cent the index lies between 41 and 54 per cent. On further calculation it was found that 25 per cent fell beyond 50 per cent. In other words, in 25 of 100 children without heart disease one is likely to find a cardiothoracic index of over 50 per cent.

In figure 1 we have gone further into the relative diagnostic value of the x-rays as compared to other methods of diagnosis. Here is

shown a comparison of the clinical diagnosis on the one hand and the x-ray and the cardiothoracic index on the other. This is not a correlation in the true statistical sense. The ordinates represent the percentages of the abnormal roentgen observations; the abscissae, the clinical diagnosis. The columns marked "all others" refer to all cases in which on the patients' first admission mitral regurgitation and stenosis with or without aortic regurgitation was diagnosed. *X* represents the diagnosis of the roentgenologist and *T* the cardiothoracic index. The columns marked *1* are the first contact, those marked *2* the end of the first year, *3*, the end of the second year and *4*, three years and later. A small number of cases that were observed for from five to seven years were included in the last columns. In the group of "early cases," on the first contact 42 per cent were diagnosed as abnormal by the roentgenologist, and 41 per cent were found to be abnormal by the measurement of the cardiothoracic index (50 per cent or over). At the end of the first year the percentages were 66 and 54, respectively; at the end of the second year they decreased slightly for both, and at the end of three years and later there was a marked increase in the roentgen diagnosis (70 per cent), but only to 46 per cent for the cardiothoracic index. From these figures it seems that the diagnosis of the general shape and size made by means of a roentgenogram taken at a distance of 6 feet is more consistent and more sensitive than the cardiothoracic index. The unexpected decrease in column *3* cannot be satisfactorily explained. In the group marked "all others" the incidence of abnormalities shown by the x-rays and the cardiothoracic indexes is almost twice as high as in the "early group" during the first year and about 50 per cent higher during the remaining periods. However, there is practically no change from year to year in this group. From column 3 it might seem that the hearts undergo a favorable change and assume a normal outline, but the explanation for the decrease in abnormal hearts indicated in this column lies rather in the fact that there were several deaths in this group. It is true, however, that the transverse diameter of the heart was not in direct proportion to the duration of the disease. In the last column the average cardiothoracic index is no greater than that in the first column. To summarize: 1. In almost one half of the early cases abnormalities were shown by the x-rays and the cardiothoracic indexes (over 50 per cent); this increased to as high as 70 per cent by the end of the third year in the group in which a diagnosis of mitral regurgitation was made. 2. The diagnosis of the form and shape of the heart by the roentgenologists is more consistent and more sensitive than the determination of the relation of the diameter of

the thorax to that of the heart, if 50 per cent is used as the limit of a normal index.

Stimulated by the work of Dr. Leo Rigler, head of the department of roentgenology at the University of Minnesota, we began to study the roentgen shadows of the barium-filled esophagus about two years ago. We have found this diagnostic procedure of great value in the differential diagnosis of difficult cases, especially in making an early diagnosis of mitral stenosis. It has been shown by a number of investigators that the esophagus is in intimate relation with the posterior surface of the heart, and that the lower portion of the esophagus is

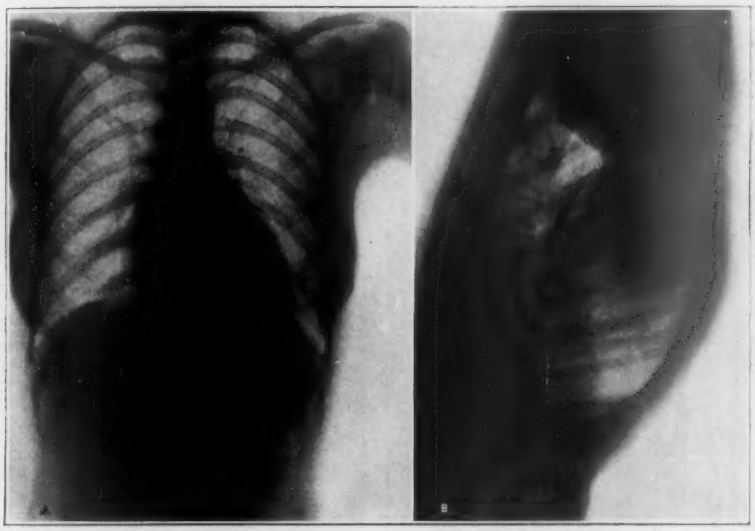

Fig. 2.—*A*, an esophagram showing the heart with mitral disease with a cardiographic index of 58 per cent; *B*, an esophagram showing compression and dislocation of the heart to the right, revealing the presence of a dilated left auricle.

in contact with the left auricle.[3] An enlargement of the left auricle may produce a compression of the esophagus that is easily detected in films that demonstrate the esophagus filled with barium. The enlargement of the auricle not only will produce a localized compression of the esophagus in its lower portion, but will usually push the esophagus posteriorly so that the space between the spine and the esophagus is diminished in this area. This is well demonstrated in figure 2. When we began using this method we had some difficulty in getting the chil-

3. Rigler, Leo: The Visualized Oesophagus in the Diagnosis of Diseases of the Heart and Aorta, Am. J. Roentgenol. **21**:563 (June) 1929; Am. Heart J. **5**:574 (June) 1930.

dren to swallow the thick barium paste that is used for adults. However, on thinning the paste somewhat and flavoring it well with cocoa or chocolate, we had no trouble in obtaining good films even of the youngest children. In the beginning we attempted to give them enough of the barium paste to fill the esophagus completely, but later we found that a heaping teaspoonful is sufficient. It is not necessary to distend the esophagus completely with the paste. One gives the child a teaspoonful of the paste, waits a few seconds and then takes the film. Undue haste in obtaining the exposure is unnecessary, as the barium paste sticks to the esophageal wall for some time after being swallowed.

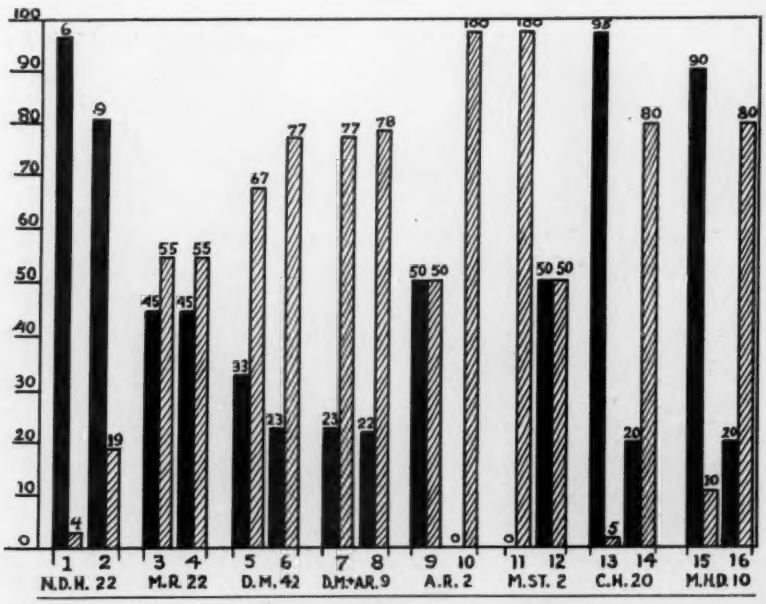

Fig. 3.—The relation in percentage of the esophagram, the roentgenologic diagnosis and the clinical diagnosis. The solid black columns represent the normal group; the striped columns, the abnormal group; the odd-numbered column, the esophagram; the even-numbered column, the roentgenogram; *N.D.H.*, no heart disease; *M.R.*, mitral regurgitation; *D.M.*, mitral regurgitation and stenosis; *D.M. and A.R.*, mitral regurgitation and stenosis and aortic regurgitation; *A.R.*, aortic regurgitation; *M.ST.*, mitral stenosis; *C.H.*, congenital heart disease; *M.H.D.*, miscellaneous heart disease.

Figure 3 represents the relation of the esophagus, the roentgen diagnosis and the clinical diagnosis. The abscissa shows the different groups of diseases. The columns marked in odd numbers represent the results obtained by esophagrams and those marked by even numbers represent the roentgenologic results. The ordinate shows the percentage of incidence. Twenty-two consecutive normal children were examined,

and in practically 100 per cent the esophagrams were normal. The one that was diagnosed as abnormal was well within the limit of experimental error. Two abnormal roentgen shadows in column 2 were also within the normal error. This method is especially valuable in differentiating certain types of children with long narrow hearts and systolic murmurs from those with organic heart disease. It is not uncommon in this type of child to obtain a postero-anterior view of the heart showing the left border straightened out, with an apparent enlargement of the conus pulmonis. It is generally believed that straightening out of the left border of the heart is diagnostic of mitral stenosis. We now know that this occurs in normal hearts, and especially in asthenic persons. Given this type of antero-posterior film with a definite loud systolic murmur at the apex, one often finds difficulty in differentiating early mitral disease from absence of heart disease. If no enlargement of the left auricle can be demonstrated by the esophagram, a diagnosis of no heart disease is made with more certainty. In the second column, we find that of twenty-two cases diagnosed as early mitral regurgitation, 55 per cent revealed abnormal esophagrams and an equal number of abnormal roentgen shadows. In other words, in this very early group, although only a systolic murmur could be heard, in more than one-half the presence of mitral stenosis was revealed by this method. Further observations of these children confirmed this fact. We have observed many children who gave a definite history of rheumatic fever or chorea and presented a systolic murmur at the apex, but in whom the roentgen evidence was doubtful. In several striking instances the esophagram showed a definite enlargement of the left auricle that was later confirmed by clinical diagnosis.

In forty-two cases of double mitral disease, 67 per cent showed abnormal esophagrams. In this group auscultation proved to be more sensitive than the esophagraphic method.

In the differential diagnosis of rheumatic heart disease and congenital heart disease the diagnosis by the esophagram is especially helpful. Of thirteen cases diagnosed as congenital heart disease, only one revealed a compressed and displaced esophagram, while 80 per cent of the same children gave evidence of enlarged hearts by plates taken at a distance of 6 feet. Not infrequently one examines a child who gives no history of having had cardiac disease from birth but who has had rheumatic fever at the age of 8 or 9 years. It is true that the location and quality of the murmur, and the marked thrill over the precordium will usually lead one to suspect a congenital defect. A marked preponderance of the electrocardiogram to the right is also supportive evidence of a congenital lesion, or the postero-anterior view may show an enlargement of the superior convexity considerably higher than is usual in mitral

stenosis. But it is often difficult to decide whether one is dealing with a congenital lesion, a rheumatic lesion or both. In such cases the esophagram will verify the diagnosis. If the postero-anterior film shows an enlargement of the heart with an enlarged pulmonary conus and if the esophagram shows no compression or displacement, the left auricle is most likely not dilated, and a diagnosis of mitral stenosis is not warranted.

Since stenosis of the mitral valve is nearly always an end-result in the progress of rheumatic heart disease, this verification of the evidence given by the roentgenogram should be resorted to early. The last column of figure 3 seems to help to localize the chamber of the heart. There were ten cases of essential hypertension in adults. They all showed extremely enlarged hearts, involving chiefly the left ventricle. In only one case of the total number was the esophagram positive, while 80 per cent of the hearts were enlarged according to the antero-posterior diagnosis. In other words, in a heart which appears much enlarged on ordinary roentgen examination, but which shows no enlargement of the left auricle by the esophagram, one can be certain that mitral disease is not the cause of the cardiac enlargement. The following case is one in point.

A boy, aged 17, was sent to the clinic by one of the school physicians because of heart trouble. There was a short distant systolic blow over the apex which disappeared on lying down and on deep inspiration. Postero-anterior films showed a large heart without a characteristic configuration. The patient did not give a history of rheumatic fever, but he knew that he had had "trouble with his heart" since the age of 8 years. It was difficult to draw any conclusions from this case. However, an esophagram showed no change in the course of the esophagus, and we were certain that there was no mitral stenosis. On further inquiry, it was found that at 8 years of age the patient had had a pericardial effusion which had been drained. We were able later to make a definite diagnosis of adhesive pericarditis.

THE ELECTROCARDIOGRAPHIC DIAGNOSIS

In a general way, it may be stated that the electrocardiogram merely gives confirmatory evidence in the diagnosis of early heart disease in children. However, one of us (Dr. Shapiro) has shown that it may be possible to diagnose early and continued rheumatic infection of the heart by frequent characteristic changes in tracings taken from time to time in children with so-called quiescent rheumatic disease.[4] As has been shown, the ambulatory patients who had rheumatic fever or chorea from three months to two years previous to examination showed abnormal changes similar to those observed by several investigators

4. Shapiro, M.: The Electrocardiograph in Subacute and Quiescent Rheumatic Fever, Am. Heart J. **5**:504 (April) 1930.

during an acute attack of rheumatic fever. Further observation will be necessary before the validity of these findings can be confirmed. Work is now in progress to determine whether or not the group of children studied two years previously, who showed these characteristic changes, actually show the progressive changes of rheumatic heart disease.

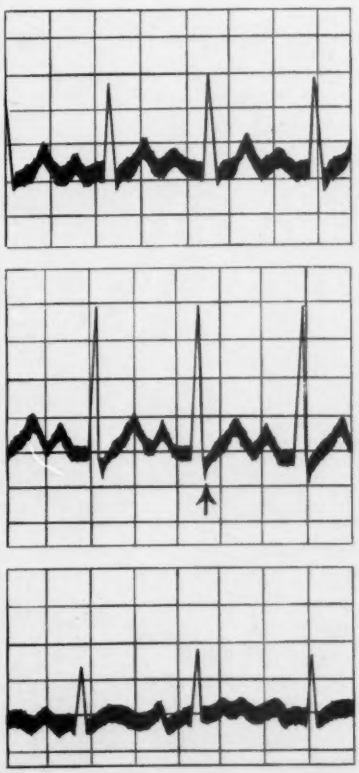

Fig. 4.—Cardiograms, showing an absent iso-electric period in lead II.

A marked right axis deviation will occasionally help in differentiating congenital heart disease from early mitral stenosis. An increase in the size and in the notching of the P wave will confirm a clinical diagnosis of mitral stenosis. The changes in the P wave resulting from an enlarged left auricle and the changes in the QRS complex, such as notching and widening or the inversion of the T wave, may indicate early myocardial disease, which cannot be diagnosed by any other method[5] (fig. 4).

5. Cohn, A. E., and Swift, H. E.: The Electrocardiogram in Rheumatic Fever, J. Exper. Med. **39**:1, 1924.

THE ETIOLOGIC DIAGNOSIS

A great deal has been written about the etiology of rheumatic heart disease. There is still a difference of opinion as to the definition of rheumatism. England seems to have set the pace for the definition of this old disease. The attitude of English writers toward rheumatism in the child is the same as that of the American writers toward tuber-

TABLE 4.—*Etiology of Rheumatic Heart Disease*

	Number of Cases	Per Cent
Rheumatic fever alone	35	39.3
Rheumatic fever +	39	43.8
Total	74	83.1
Tonsillitis alone	7	7.8
Tonsillitis +	19	21.4
Total	26	29.2
Chorea alone	3	3.3
Chorea +	15	16.8
Total	18	20.1
Growing pains alone	2	2.3
Growing pains +	13	14.5
Total	15	16.8
Scarlet fever alone	2	2.3
Scarlet fever +	11	11.4
Total	13	14.7

	No. of Cases*		No. of Cases*		No. of Cases*
R	35	R + G + T	2	T + C	1
T	7	R + G + C	4	T + R	10
C	3	R + S + T	3	T + G	1
G	2				
S	2	R + S + T + G	1	C + R	8
		R + S + T + C	1	C + T	1
R + T	10			C + G	1
R + S	6	G + R	4		
R + C	8	G + T	1	S + R	6
R + G	4	G + C	1		

* R indicates rheumatic fever; T, tonsillitis; C, chorea; G, growing pains; S, scarlet fever.

culosis. Because it is so prevalent in their country and because it is so serious a disease, it is possible that they have taken the attitude of the specialist who suspects first the disease in which he has specialized.[6]

6. Poynton, F. J., and Askins, R. A.: Rheumatic Infection in Childhood, Brit. M. J. **2**:788 (Oct. 31) 1925. Poynton, F. J., in Garrod: Diseases of Children, New York, William Wood & Company, 1929, chapt. 18; Cardiac Infections in Children, Brit. M. J. **2**:986 (Nov. 29) 1924. Miller, R.: Rheumatic Fever, Brit. M. J. (supp.) **2**:5 and 16 (July 3) 1926. Coombs, C. F.: Rheumatic Heart Disease, New York, William Wood & Company, 1924.

Table 4 represents an etiologic analysis of the seven most commonly considered causes of rheumatic heart disease. The following special questionnaire [7] was used for that purpose. It is natural that with a difference in the material and different definitions in mind, the statistics will be different in different clinics.

PUPIL'S MEDICAL HISTORY — Minneapolis Public Schools
Heart Clinic — Hygiene Department

Date .....
.....
Name ..... Nationality .....
Address ..... School ....
Sex ..... Date of birth ..... Grade .....
Father's occupation .....
Number of children in family .....
How many are older than this child? .....
Have tonsils been removed? .....
If so, when? ..... Month ..... Year .....
Frequent tonsillitis—Yes ..... No .....
Scarlet Fever ..... Month ..... Year .....
Inflammatory Rheumatism (Rheumatic Fever) Yes ..... No .....
How long sick in bed? ..... Did doctor say child had rheumatism? .....
First attack .....Month.....Year
Second attack .....Month.....Year
Third attack .....Month.....Year
St. Vitus Dance—Yes..... No .....
How long sick in bed?..... Did doctor say child had St. Vitus Dance?.....
First attack .....Month.....Year
Second attack .....Month.....Year
Third attack .....Month.....Year
Fourth attack .....Month.....Year
Growing Pains—Yes..... No .....
If so, Year .....
What other sickness has child had?.....
Family History:
Has anyone in the family had:

	Inflammatory Rheumatism	St. Vitus Dance	Heart Disease
Father	.....	.....	.....
Mother	.....	.....	.....
Sister	.....	.....	.....
Brother	.....	.....	.....
	.....	.....	

Rheumatic fever alone is responsible for 39.3 per cent of heart disease, but in combination with all of the other forms of rheumatism it is present in 83.1 per cent of all cases. Chorea and scarlet fever alone are present in a small number, but in combination they represent

7. The questionnaire was devised by Gertrude Shapiro.

one fifth and one sixth of the total number. Other writers have found that growing pains alone are responsible for from 10 to 25 per cent of rheumatic heart disease. We have found, however, a much smaller incidence of growing pains as the only cause. With other conditions, growing pains are the etiologic factor in almost 17 per cent of our series. At the bottom of table 4 are shown the various combinations of rheumatism with other conditions.

Functional Diagnosis.—During and since the war a great deal of attention has been given to functional tests of the heart. All of these tests were used in order to determine the reactions of the pulse rate

TABLE 5.—*A Comparison of Normal Children and Those with Heart Disease in Exercise with Dumbbells*

Normal							Heart Disease						
	Blood Pressure				Pulse Rate			Blood Pressure				Pulse Rate	
Age, Years	Maximum Rise, Mm.	Time Maximum Reading, Sec.	Percentage of Recovery at 120 Sec.	Percentage of Recovery at 180 Sec.	Percentage of Recovery at 120 Sec.	Percentage of Recovery at 180 Sec.	Age, Years	Maximum Rise, Mm.	Time Maximum Reading, Sec.	Percentage of Recovery at 120 Sec.	Percentage of Recovery at 180 Sec.	Percentage of Recovery at 120 Sec.	Percentage of Recovery at 180 Sec.
14	39	45	79.1	86.6	100.0	100.0	15	47	15	84.3	94.1	100.0	100.0
14	35	30	88.6	93.5	100.0	100.0	14	30	35	82.8	86.5	81.9	90.0
13	35	55	76.3	86.9	.....	87.5	14	24	40	100.0	100.0	100.0	100.0
13	46	55	77.3	86.8	100.0	100.0	14	28	40	95.0	96.2	100.0	100.0
13	35	45	79.0	82.8	100.0	100.0	13	33	30	86.5	95.7	.....	92.0
13	40	35	94.5	100.0	100.0	100.0	13	47	40	83.6	93.1	91.0	91.0
12	35	25	85.4	90.8	87.5	91.8	13	41	40	81.7	89.2	79.2	91.6
12	37	35	86.6	94.2	85.7	90.0	12	27	30	81.7	86.8	.....	100.0
12	35	35	86.5	87.2	.....	80.0	12	65	10	81.5	82.6	100.0	100.0
11	32	30	87.9	96.3	.....	100.0	12	30	20	95.8	95.8	83.4	83.4
10	19	35	87.8	93.1	100.0	100.0	12	49	65	73.3	82.1	100.0	100.0
10	43	35	71.6	93.0	100.0	100.0	10	30	20	95.4	100.0	100.0	100.0
9	37	25	86.9	96.3	.....	.....	8	41	20	87.9	92.3	80.0	88.9
Aver.	41	39.4	83.4	91.3	97.0	95.5	Aver.	37.8	31.1	85.3	91.9	91.0	95.1

and blood pressure to change of posture and after measured amounts of exercise. The Barringer, Schneider, Crampton and Graupner tests are examples. In 1923 we studied this subject in thirteen normal children and in an equal number of children who were suffering from rheumatic heart disease (table 5).[8]

The exercise consisted of thirty-five full length swings with dumbbells weighing 4 pounds (1,814 Gm.) (only one child, 8 years of age, swung a pair of dumbbells weighing 2 pounds [907 Gm.]). The following items are tabulated in table 5: (1) the maximum rise and

8. Seham, M., and Egerer-Seham, G.: Physiology of Exercise; Part 3. An Investigation of Cardiovascular Tests in Normal Children and in Children with Tuberculosis and Valvular Heart Disease, Am. J. Dis. Child **26**:554 (Dec.) 1923.

the time of maximum reading of the blood pressure (columns 1 and 2); (2) the percentage recovery of the blood pressure at 120 seconds and 180 seconds, respectively (column 3), and (3) the percentage recovery of the pulse rate after the same periods (column 4).

Comparing the results obtained in normal children with those obtained in children with heart disease, one is impressed by their similarity. The maximum rise of the blood pressure in children with heart disease is about 3 mm. lower than that in normal children. This result certainly lies within the limit of error for readings of the blood pressure. The time of maximum readings is slightly lower in persons with heart disease, 31.1 as against 39.4 seconds in normal subjects. The percentage of recovery of the blood pressure amounts to 83.4 and 91.3, respectively, in normal children, and 85.3 and 91.9, respectively, for those with heart disease. This difference, however, cannot be considered sufficient to be of practical value in diagnosing myocardial insufficiency.

During the past two years we have discarded these functional tests and have employed the simple method of taking the history carefully, laying special emphasis on the child's reactions to the demands made on him in his everyday work. We have used the functional classification recommended by the American Heart Association. The statistical study of the relation of the functional diagnosis to the clinical and the roentgen diagnosis was not satisfactory, and therefore we do not present the tables at this time. In general, however, the second degree cases representing mild signs of decompensation were present far more frequently in those labeled "all others" than in the early stages.

The correlation of the roentgen diagnosis and the functional diagnosis was low, but there was a suggestion that the more abnormal the cardiothoracic index, the more frequent were the signs of decompensation. However, there were so many exceptions that in the individual case it is doubtful whether this method will ever prove to be of diagnostic value. It may take years after the muscle of the heart becomes dilated and hypertrophied before any functional signs and symptoms become apparent. On the other hand, signs and symptoms that simulate organic heart disease may be present in many children who do not have heart disease. The functional method is the least reliable of all the methods of differential diagnosis.

SUMMARY

Rheumatic heart disease is so extremely variable in its virulence and progress that it is almost impossible to formulate a single scheme or to apply any one method of early diagnosis that can be depended on with any degree of constancy.

One child may suffer from decompensation of the heart from a first attack of rheumatism while in bed; another may have repeated attacks of rheumatism and escape involvement of the heart. In one case definite signs and symptoms of organic heart disease may become apparent in a very short time, while in another it may be one or two years after the first attack of acute endocarditis before the heart has undergone sufficient progressive changes to warrant the diagnosis of organic heart disease.

CONCLUSION

1. In 809 patients admitted during a period of seven years, 46 per cent of the cases were diagnosed as organic heart disease and 54 per cent as "no heart disease."

2. Of 379 cases of organic heart disease, 18 per cent were congenital lesions of the heart, 74 per cent were rheumatic in origin, only 3.6 per cent were due to other causes and 4.4 per cent were undiagnosed.

3. For early diagnosis of rheumatic heart disease, all available methods are necessary, namely: (1) etiologic, (2) structural, (3) functional, (4) roentgenologic and esophagraphic and (5) electrocardiographic.

4. The problem of differential diagnosis revolves chiefly around the children who present the following conditions: (1) a systolic murmur, with or without circulatory signs and symptoms, (2) rheumatism and a systolic murmur that has escaped carditis, (3) rheumatism and a systolic murmur that ultimately develops into chronic endocarditis and (4) congenital heart disease, especially patency.

5. According to our statistics, the most reliable means of diagnosis is the history. A questionnaire gives more positive data than informal questioning. In 92 per cent of the cases of early mitral disease we were able to obtain a history of rheumatism according to the definition that we have used.

6. Rheumatism alone causes 39.3 per cent of rheumatic heart disease; rheumatism in combination with other causes, 83.1 per cent. Growing pains alone are responsible for 2.3 per cent.

7. In the first contact, 42 per cent of the cases diagnosed as "early mitral disease" show abnormalities revealed by the x-rays; in the cases called "all others," 84 per cent were abnormal.

8. The roentgenogram taken at a distance of 6 feet is as reliable for the practical purposes of diagnosis in children as the orthodiagram.

9. The 50 per cent cardiothoracic index that is used as a normal standard is statistically not highly reliable. In 66 per cent of normal children the cardiothoracic index is between 44 and 50 per cent, and

the remaining 33 per cent fall between 41 and 54 per cent. In 25 of 100 consecutive examinations of normal children, one is likely to find a cardiothoracic index of over 50 per cent.

10. A positive esophagram reveals a dilatation of the left auricle. This does not appear in children with congenital heart disease, in those with miscellaneous heart disease or in normal children.

11. The esophagraphic results are positive in 55 per cent in cases diagnosed as mitral regurgitation. In this group the esophagram is more sensitive than auscultation. It gives positive observations in 67 per cent in the group diagnosed as double mitral disease and 77 per cent in those with double mitral disease and aortic regurgitation.

12. The absent iso-electric period in lead II and the delayed P-R interval are indications of active rheumatic infection. The former does not occur when there has been no rheumatism.

13. The so-called functional heart tests are of no value in determining the myocardial sufficiency of the heart.

14. The functional method of diagnosis by the taking of the patient's history is probably the least sensitive of all methods for a diagnosis in the early stages.

This study was made possible by a grant from the University of Minnesota Medical School.

Dr. M. Van Wagenen and Dr. Grete Seham gave us help in the statistical analysis, Dr. Leo Rigler and Dr. Walter Ude gave their time and interest in behalf of the roentgen study, and Dr. M. Mathanson read and criticized the paper.

ENCEPHALOGRAPHY IN CHILDREN *

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The roentgenographic demonstration of air accidentally introduced into the skull in fractures served as the basis of subsequent investigation by artificial insufflation. The first and foremost step was made by Dandy,[1] who sought a method of visualizing tumors of the brain. In 1918, he successfully used air for ventriculography in a series of twenty cases, and in 1919, he obtained satisfactory visualization by lumbar injection.[2] Two years later, Wideroe [3] and Bingel [4] independently used lumbar injection to demonstrate the obstruction caused by tumors of the spinal cord. They also found these studies valuable in visualizing the ventricles, basal cisterns and subarachnoid cortical spaces.

During subsequent years, encephalography was further substantiated by its correlation with findings at operation and autopsy (Kruse [5]), by the recognition of its dangers and complications (Koschewnikow [6] and

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* The observations recorded in this paper were completed in 1929 and reported before the Brooklyn Pediatric Society, April 23, 1930. At the last meeting of the American Pediatric Society, one of us summarized this work in a discussion of Dr. Crothers' paper. This communication represents the detailed report on which that discussion was based.

1. Dandy, W. E.: Ventriculography Following the Injection of Air into the Cerebral Ventricles, Ann. Surg. **68**:5, 1918.

2. Dandy, W. E.: Roentgenography of the Brain After the Injection of Air into the Spinal Canal, Ann. Surg. **70**:397, 1919.

3. Wideroe, S.: Ueber die diagnostische Bedeutung der intraspinalen Luftinjectionen bei Rückenmarksleiden, besonders bei Geschwülsten, Zentralbl. f. Chir. **48**:394, 1921.

4. Bingel, A.: Encephalographie, eine Methode zur roentgenographischen Darstellung des Gehirns, Fortschr. a. d. Geb. d. Röntgenstrahlen **28**:205, 1921; Neben und Nachwirkungen bei Gaseinblasungen in den Lumbalkanal (therapeutische Möglichkeiten und Erfahrungen), Deutsche Ztschr. f. Nervenh. **75**:230, 1922.

5. Kruse, F.: Cerebrale Krankheiten des Kindesalters im typischen Encephalogrammen, Ergebn. d. inn. Med. u. Kinderh. **37**:332, 1930.

6. Koschewnikow, A. N.: Subjective und objective Ergebnisse einer encephalographische Untersuchung, Ztschr. f. d. ges. Neurol. u. Psychiat. **104**:374, 1926.

Grant [7]), by its revelation of a vast museum of pathologico-anatomic conditions in the living (Foerster [8] and Wartenburg [9]) and by the realization of its therapeutic value in some neurologic conditions (Eckstein,[10] Sharp [11] and Penfield [12]). Thus, a rich literature on encephalography developed independently in Germany and America, completely reviewed by Kruse [5] in his recent monograph on that subject. The diagnostic and prognostic value of the procedure in pediatrics was stressed in this country by Crothers [13] in a study of three hundred patients with fixed lesions of the brain.

METHOD

Atmospheric air has generally proved most satisfactory for encephalography and has supplanted all other mediums. Heating, sterilization and filtration of the air are unnecessary complications of an ordinarily simple technic.

Though various types of apparatus have been introduced, all intended to maintain constant pressure, it appears that Dandy's original simple syringe and needle suit the purpose admirably. Bingel [4] advocated the puncture of two adjacent spaces, the upper for the influx of air under manometric control and the lower for the escape of fluid. A miniature Potain outfit has been employed to insure replacement by equal volume. Pancoast and Fay [14] emphasized vehemently the need for restricting the pressure in the circuit below 20 mm. of mercury. Though Kruse recorded the pressure of the spinal fluid during the procedure, he believed that its importance has been overestimated. Crothers expressed skepticism concerning the value of spinal manometry in children. The task of standardization of both the operative procedure and the roentgenographic technic has been taken up by the Committee on Standardization of Encephalography and has recently

7. Grant, F.: Ventriculography, Am. J. Roentgenol. **18**:264, 1927.

8. Foerster, O.: Encephalographische Erfahrungen, Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:512, 1925.

9. Wartenburg, R.: Beitrag zur Encephalographie und Myelographie, Arch. f. Psychiat. **77**:507, 1926.

10. Eckstein, A.: Die encephalographische Darstellung der Ventrikel im Kindesalter, Ergebn. d. inn. Med. u. Kinderh. **32**:531, 1927.

11. Sharp, E. A.: Artificial Pneumorachis in the Treatment of Acute Infections of the Meninges, Arch. Neurol. & Psychiat. **6**:669 (Dec.) 1921.

12. Penfield, W.: Chronic Meningeal (Post-Traumatic) Headache and Its Specific Treatment by Lumbar Air Insufflation, Surg., Gynec. & Obst. **45**:747, 1927.

13. Crothers, B.; Vogt, E. C., and Eley, R. C.: Encephalography in Cases of Fixed Lesions of the Brain, Am. J. Dis. Child. **40**:227 (Aug.) 1930.

14. Pancoast, H. K., and Fay, T.: Encephalography: Roentgenological and Clinical Considerations for Its Use, Am. J. Roentgenol. **21**:421, 1929.

been outlined by Pendergrass.[15] To avoid disturbing the spinal needle by repeated adjustment and removal of the syringe during the procedure, we have found it advisable to use a three-way stopcock, connected by small pieces of rubber tubing to needle and syringe. The flexible rubber tubing allows for manipulation without displacement of the needle (fig. 1).

The details of technic are essentially those described by Crothers.

Each child is subjected to a complete neurologic and clinical investigation before encephalography is attempted. Some authors advocate the use of a general anesthetic, but in most clinics satisfactory results have been obtained with sedatives. In most instances, preliminary injections of morphine and atropine have sufficed. Epileptic patients should be given phenobarbital for long periods preceding encephalography.

With the child in the reclining position, a lumbar puncture is performed, and a small amount of fluid taken for examination. The patient is then raised to a sitting position, the body bent slightly forward and the head firmly supported

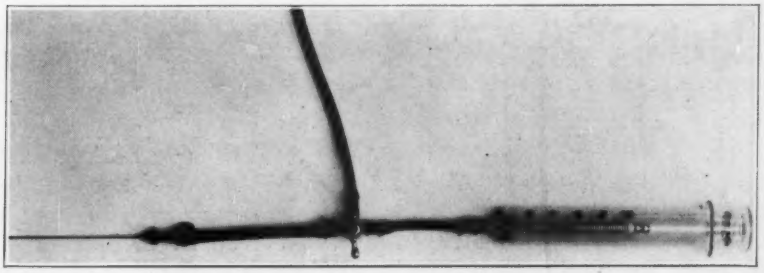

Fig. 1.—Apparatus for replacement with air in encephalography.

by an assistant, and replacement with air is begun. For each 10 cc. of fluid removed, from 8 to 9 cc. of air is injected, to allow for expansion within the cranial cavity as the temperature of the air rises from that of the room to that of the body. If a manometer is to be used, pressures can be taken with each exchange of air and fluid. Should difficulty in the flow be encountered, it may be necessary to rotate the needle or to inject a small amount of air. A slight haze from a drop of blood in the puncture tract should make one cautious during the subsequent injection, but a frankly hemorrhagic fluid or evidences of increased intracranial pressure call for immediate cessation of the procedure (Kruse[5]). After the flow has completely ceased, gentle rotation of the head from side to side may serve to release fluid trapped in the ventricles and basal cisterns, thus accomplishing more complete and symmetrical filling (Kruse).

How far to carry the procedure and how much air is necessary to effect a satisfactory representation cannot be stated as a generalization. Whereas 20 cc. may be of value in one case, it may be necessary in another to inject 400 cc. before adequate visualization takes place. It

15. Pendergrass, E. P.: Interpretation of Encephalographic Observations, Arch. Neurol. & Psychiat. **23**:946 (May) 1930.

is advisable to strive for as complete removal of fluid as possible, though it seems unlikely that one can empty all the available fluid from the cerebrospinal system, small quantities always remaining in the inferior horn of the lateral ventricles, the third ventricle and the cortical sulci. With the injection of from 60 to 120 cc. of air, adequate visualization can be effected in most instances. In a case of encephalitis following varicella, encephalography was attempted by the lumbar and suboccipital routes, but only 30 cc. of fluid could be withdrawn in each instance. When a similar amount of air was injected only the cortical markings were portrayed, and the air was evidently unable to enter the ventricles. Our neurologist, Dr. I. J. Sands, suggested that the cerebral foramina were being temporarily blocked by reactionary edema which might effectively be reduced by cerebral dehydration. This was accomplished by intravenous injections of hypertonic solutions of dextrose

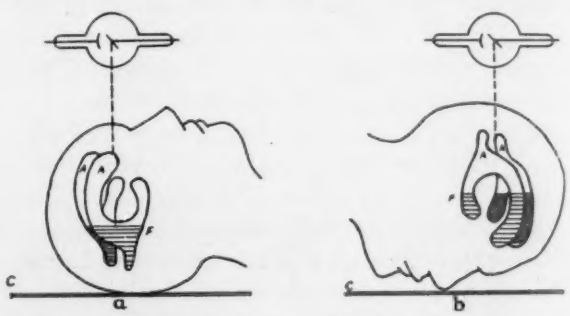

Fig. 2.—*a*, position of patient for fronto-occipital exposure (see fig. 4*A*): *A*, air; *C*, film cassette; *F*, fluid level. *b*, position of patient for occipitofrontal exposure (see fig. 4*C*).

and magnesium sulphate. At subsequent encephalography, 130 cc. of spinal fluid was removed and about 120 cc. of air injected. Normal ventricles were visualized.

Contrary to Eckstein's exhortations to put the patient to bed for several hours after the procedure, thereby preventing symptoms of collapse, we have found no ill effects from having the roentgen investigation started immediately. Because air is quickly absorbed from the cortical spaces, as shown by Weigeld,[16] and because production of fluid in the ventricles goes on at a rapid rate, one must make roentgen exposures immediately after the injection of air is completed in order to obtain the most detailed studies. The patient's condition permitting, one starts the roentgen investigation best with fluoroscopy. Though the finer details are not recognizable, the intermediation of this procedure

16. Weigeld, W.: Die Bedeutung der Lufteinblasung für Hirn und Rückenmarkdiagnostik, Deutsche Ztschr. f. Nervenh. **77**:165, 1923.

gives valuable information as to the degree, absence or asymmetry of the filling, and can frequently spare an entire series of exposures by showing an unsatisfactorily filled hydrocephalus. By manipulation of the patient's head in some cases, one may effect better distribution of air in the fluid pathways. A clearer tridimensional concept of the ventricular system may also be obtained in this fashion and the various shadows traced to their counterparts in the four views. Stereoscopic exposures, because of their value in spatial localization, have been made part of the routine roentgenography by the Committee on Standardization of Encephalography.[15]

REACTION TO THE PROCEDURE

The effects of encephalography on the patient's condition merit careful consideration. It was previously believed that the replacement of 100 cc. of fluid by air would lead to instant death, but experience has demonstrated that such procedures are without harmful consequences. However, great care in the performance of the entire procedure is indicated in order to minimize sequelae. Headache may accompany the entrance of air into the ventricles. Vomiting, shallow respiration, perspiration and pallor occasionally appear. During the escape of fluid, bradycardia sets in, and it disappears with insufflation of air. Severe collapse is rarely observed. Increasing drowsiness sets in toward the close of the procedure and persists for several hours, so that one may really speak of an air anesthesia. Several hours after the procedure the temperature usually rises to 100.5 F., occasionally higher, and gradually falls to normal. In isolated cases meningitic symptoms may supervene, the spinal fluid showing evidences of an aseptic meningitis. Crothers has usually found a positive globulin reaction in the spinal fluid following encephalography even when the cell count is not markedly increased. The cellular reaction is in the nature of a polymorphonucleosis, ranging from 50 to 98 per cent. Friedman,[17] in a review of 47 cases, reported an instance in which the cell count rose to 19,500 cells per cubic millimeter. In our experience the greatest cellular reaction was one of 8,000 cells, 89 per cent of which were lymphocytes. The absence of organisms, the return of the cell count to normal and the disappearance of the meningitic symptoms within forty-eight hours are evidences that the meningitis was aseptic.

The reactions in individual cases are very diverse, depending on the underlying conditions. Bingel[4] found that in certain conditions the brain tissue appears to be insensible, since patients with cerebral syphilis,

17. Friedman, E. D.; Snow, W., and Kasanin, J.: Experiences with Encephalography via the Lumbar Route, Arch. Neurol. & Psychiat. **19**:762 (May) 1928.

epilepsy and poliomyelitis endure the procedure unusually well. As a rule, younger children tolerate the procedure well, frequently sitting up and playing in bed several hours after the injection. In any case, these reactions subside within four days. We have never observed the recrudescence of a receding meningitis. The headache encountered responds favorably to such coal tar derivatives as antipyrine and acetphenetidin. In hydrocephalic persons in whom a reactionary hypersecretion has supervened, intravenous hypertonic solutions and repeated lumbar punctures have been of value in relieving the symptoms.

Fatalities have been reported by Fay and Heidrich. The mortality in ventriculography was 12.3 per cent in 519 cases collected by Heidrich, caused largely by inoperable tumors of the brain and diffuse gliomas. In these, encephalography is contraindicated. Heidrich,[18] in 1927, calculated a mortality of less than 0.67 per cent in 895 cases, and Pancoast and Fay,[14] in 1929, 1.2 per cent in 1,529 cases.

It is gratifying that encephalography frequently exerts a favorable therapeutic influence on the course of a disease. It has been shown to have considerable value in epidemic meningitis in that it lessens the tendency to the formation of adhesions. Amelioration of symptoms in epilepsy for several weeks or months is not infrequent (Friedman[19] and Bingel[4]). Recalcitrant, as well as posttraumatic, headaches have yielded to insufflation of air (Penfield[12]). In a case of Little's disease, the disappearance of spasticity for two weeks, during which time air had remained trapped in the ventricles, was reported by Kruse.[5] Eckstein[10] reported symptomatic improvement in cases of tuberculous meningitis. Finkelstein[20] successfully treated patients for meningococcus meningitis with injections of air only. In acute and chronic encephalitis, on the other hand, aggravation of symptoms has occasionally been noted.

INTERPRETATION

An understanding of the anatomy and physiology of the channels of the cerebrospinal fluid is essential for the proper interpretation of the roentgenograms (fig. 3). The fluid is secreted by the choroid plexus, located on the floor of the lateral ventricles, each of which is composed of a vestibule and an anterior, posterior and inferior horn. The fluid then passes through the paired foramina of Monro into the third ventricle, which is situated in the midline, in the hollow between the anterior

18. Heidrich, L.: Die Encephalographie und Ventriculographie, Ergebn. d. Chir. u. Orthop. **20**:156, 1927.

19. Friedman, A.: Therapeutische Möglichkeiten und Ergebnisse der Lufteinblassung in die Liquorraume von Gehirn und Rückenmark, Deutsche Ztschr. f. Nervenh. **106**:82, 1928.

20. Finkelstein, H.: Personal communication to the authors.

and inferior horns. Passing through the aqueduct of Sylvius into the fourth ventricle, the fluid emerges by way of the foramina of Magendie and Luschka into the basal cisterns. A small part of the fluid passes down the spinal canal and is probably absorbed along the perineural lymphatics, whereas the greater portion traverses the cortical sulci to be absorbed by the pacchionian bodies. Obstruction at any point would cause distention in the ventricular system behind it. Cortical shrinkage, from whatever cause, leads to a compensatory increase in the volume of fluid covering the surface of the brain.

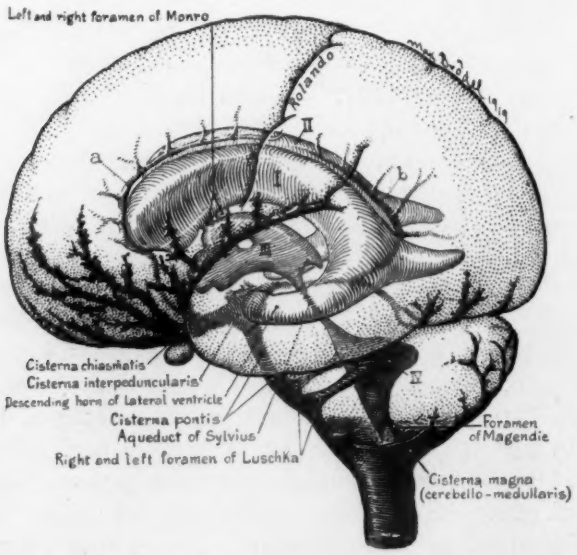

Fig. 3.—Sketch of the anatomy of the ventricular system.

Our present concept of the normal encephalogram has been derived from numerous observations on children presenting no abnormal neurologic findings, from a comparison of roentgenograms with sketches of craniocerebral topography (Schuster [21]) and from experimental injections of barium sulphate in cadavers (Winkler [22]). The fronto-occipital view (fig. 4 *A*) presents a butterfly figure projected by the anterior horns of the lateral ventricles. With good replacement by air, the third ventricle is also visualized in this exposure, appearing as a lentil-shaped

21. Schuster, J.: Ueber die Verschiedenheit der Bilder bei Weiderholter encephalographischen Untersuchung des Gehirns, Arch. f. Psychiat. **77**:532, 1926; Einiges zur roentgenographischen Darstellung des Gehirns, Deutsche Ztschr. f. Nervenh. **80**:129, 1923.

22. Winkler, H.: Beiträge zur Hirnanatomie im Encephalogramm, Deutsche Ztschr. f. Nervenh. **99**:277, 1927.

structure in the midline. The basal ganglions form the lateral walls of the midportions of the anterior horns. The choroid plexus may sometimes be visible by virtue of its encroachment on the air shadow. With maximum replacement by air, one may visualize the inferior horns as curvilinear appendages to the lateral aspect of the "butterfly" especially when these are dilated.

In the occipito-frontal exposure (figs. 4 *B* and *C*), one visualizes the posterior and inferior horns which form a figure described as ram's

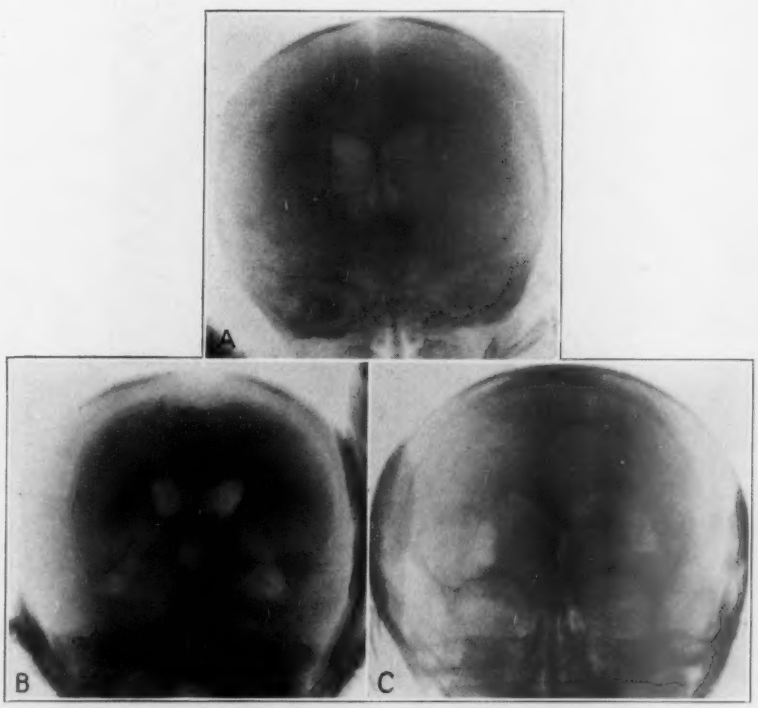

Fig. 4.—*A*, encephalogram showing normal ventricles, fronto-occipital view: butterfly figures. *B*, encephalogram showing normal ventricles. Note lateral ventricles, inferior and posterior horns, and third ventricles, occipitofrontal view. *C*, encephalogram showing normal ventricles, occipitofrontal view. Note figure of "ram's horns."

horns. Tumors of the cerebellum and parietal lobes may manifest themselves by encroachment on, or displacement of these horns. Dilatation of the horns occurs on the homolateral side in vascular, focal, epileptic or traumatic lesions, as well as in congenital malformations and infantile birth palsies. Asymmetrical and symmetrical dilatation of the ventricles is best observed in the first two views. Unilateral cortical shrinkage is well portrayed in either of these two exposures.

The lateral views (fig. 5 *A*) are most effective in a portrayal of the configuration of the entire ventricular system, basal cisterns and cortical markings, though they are not as effective in depicting displacement. On a satisfactory plate, all three horns of each lateral ventricle and the foramen of Monro, third ventricle (and its pineal, interpeduncular and chiasmatic recesses), aqueduct of Sylvius, fourth ventricle and cisterna magna, pontis, interpeduncularis and chiasmatis may be visualized. The cortical markings are normally more prominent over the frontal

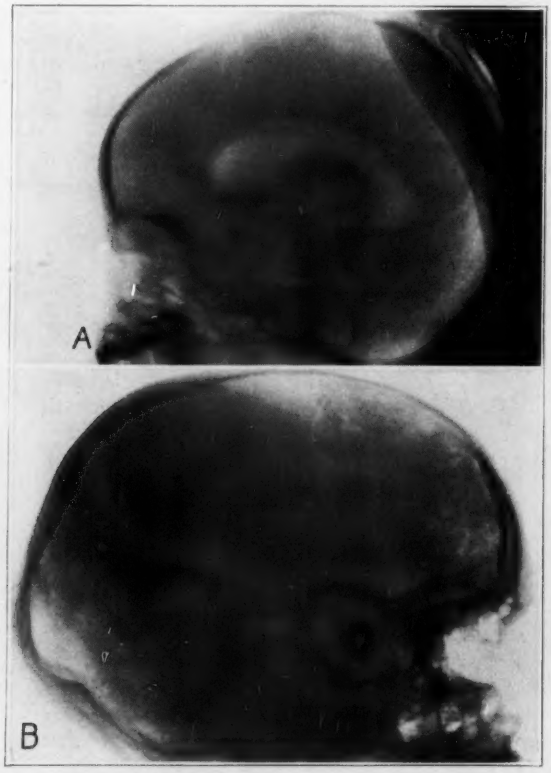

Fig. 5.—*A*, roentgenogram of the lateral view. Note outline of entire ventricular system. Compare with figure *3*. *B*, encephalogram of child showing well defined cortical markings.

and temporal lobes than over the occipital (fig. 5 *B*). In cases of communicating hydrocephalus, the basal cisterns and cortical markings appear much more prominently, while in instances of hemorrhage, meningitis and arachnoiditis the normal pathways may be obliterated or diminished, with compensatory enlargement of accessory channels. A more detailed interpretation of the roentgenograms has been reported by Pendergrass [15] who carefully indicated all the changes in cortical markings, ventricles and cisterns and pointed out their significance.

Encephalography serves a somewhat different purpose in children from that in adults. Whereas in the latter its greatest value lies in the localization of tumors of the brain, this condition rarely exists in children. In the latter it is of great value in depicting pathologic intracranial conditions in infantile birth palsies, congenital malformations of the brain (such as microcephalia and porencephalia), congenital internal hydrocephalus and tuberculous and purulent meningitides, all of which are less frequently found in adults.

REPORT OF CASES

In the pediatric wards of the Jewish Hospital during the year 1929, encephalograms were made in 20 cases presenting the following diagnoses: Meningococcus meningitis, 4 cases; cerebral maldevelopment, 5; intracranial birth injury with hydrocephalus, 2; abscess of the brain, 2; epilepsy, 2; encephalitis, 2; tuberculous meningitis, 1; tumor of the brain (unverified), 1, and congenital communicating hydrocephalus, 1.

In three of the cases of meningococcus meningitis, in which encephalography was performed during the recession of the infection, we encountered normal encephalograms. Mild reactions followed the procedure in two of the cases; these showed a slight increase in the cell count of the spinal fluid, vomiting and elevation of temperature to 102 F., all of which disappeared in three days. The third patient had no reaction except for the usual drowsiness, and all three were discharged from the hospital as recovered. The fourth patient (case 5) of this group showed marked generalized ventricular dilatation, with no subsequent reaction and recovered completely; apparently this was a temporary hydrocephalus.

All five cases of cerebral maldevelopment demonstrated intracranial disease, in vivo, on the roentgenogram. One showed a definite porencephalia on the right side, which appeared as a large irregular air shadow near the temporal region (case 6). This child suffered only a mild elevation of temperature for two days after the procedure. Another manifestated hemiatrophy of the cortical brain substance and irregular deformity of the ventricles. This patient's reaction to the procedure was severe in that he became cyanotic, with shallow respirations, and required stimulation. His temperature was elevated for two days, reaching 104 F. (case 2). The third case was one of microcephalia and status epilepticus, showing deformity of the skull and ventricles. A severe reaction followed the procedure in this instance: stupor and elevation of temperature appeared with cessation of respiration thirty hours after the insufflation. Postmortem examination revealed subarachnoid hemorrhage (case 1). The other two cases showed evidence

of excessive cortical markings incident to shrinkage of the brain with porencephalia of the frontal lobe. Both patients reacted only with mild elevation of temperature.

Both our cases of birth injury showed mild hydrocephalus with only moderate reaction to encephalography. One of these patients, who even at 1 year of age lacked visual perception of light or objects, revealed marked enlargement of the posterior horns suggesting probable involvement of the occipital lobes (case 4). The other revealed an absence of ventricular filling though cortical markings were present.

One of the patients with abscess of the brain showed deformity of the homolateral ventricle with definite displacement of both ventricles to the opposite side. She reacted with vomiting, severe headache and mild elevation of temperature for one day. A subsequent operation by Dandy corroborated the clinical diagnosis and encephalographic finding of right-sided precentral abscess of the brain. Following drainage, the patient made an uneventful recovery (case 6). The other case, one of bilateral abscess of the frontal lobe, secondary to sinusitis, was not diagnosed until postmortem examination.

The case of tuberculous meningitis manifested slight generalized hydrocephalus with no subsequent reaction. Our epileptic patients showed normal encephalograms with no appreciable changes in their condition following the procedure. The same was true of the cases of encephalitis. One patient in whose case a questionable diagnosis of tumor of the brain had been made revealed nothing abnormal on the encephalogram and had no immediate reaction, but two days later suddenly died in a convulsive state. No postmortem examination was made.

The following reports are summaries of some of the cases referred to.

CASE 1.—B. G., a white girl, aged 4½ years, was admitted to the Jewish Hospital on Nov. 29, 1929, with the complaint of generalized convulsions since 6 months of age. The delivery had been precipitate with no evident sequelae, and the child had progressed normally until the age of 6 months. Thereafter her mental development had remained stationary, although her physical growth continued somewhat slowly. At 6 months she had a generalized convulsion, tonic in nature, with laryngospasm and frothing at the mouth. Such seizures recurred from five to ten times an hour for periods of from one to four weeks and less frequently between these periods. At the time of admission she had had a practically continuous series of seizures for five days.

The child was malnourished and maldeveloped. Her head measured 3¾ inches (9.5 cm.) less in circumference than her chest. The eyegrounds were normal. There was hypotonicity of all the muscles, continuous purposeless incoordinate movements of the extremities and frequent recurrent convulsive seizures with rigidity and hyperextension, laryngospasm, cyanosis and frothing at the mouth. The clinical diagnosis was microcephalia, cerebral maldevelopment and status epilepticus.

Encephalography.—On the fourth day in the hospital encephalography was performed; 40 cc. of fluid was removed, and 35 cc. of air injected. The spinal fluid was normal. The temperature rose to 103 F. the following day, with the onset of stupor and shallow respirations until the child died thirty-six hours after the injection of air. The encephalogram (fig. 6 *A*) showed evident distortion of the skull paralleled by similar distortion of the lateral ventricles. The third ventricle, which was faintly visualized, was somewhat distorted. Air was present in small amounts in the cortical spaces.

The impression was that the case was one of microcephalia and cerebral maldevelopment.

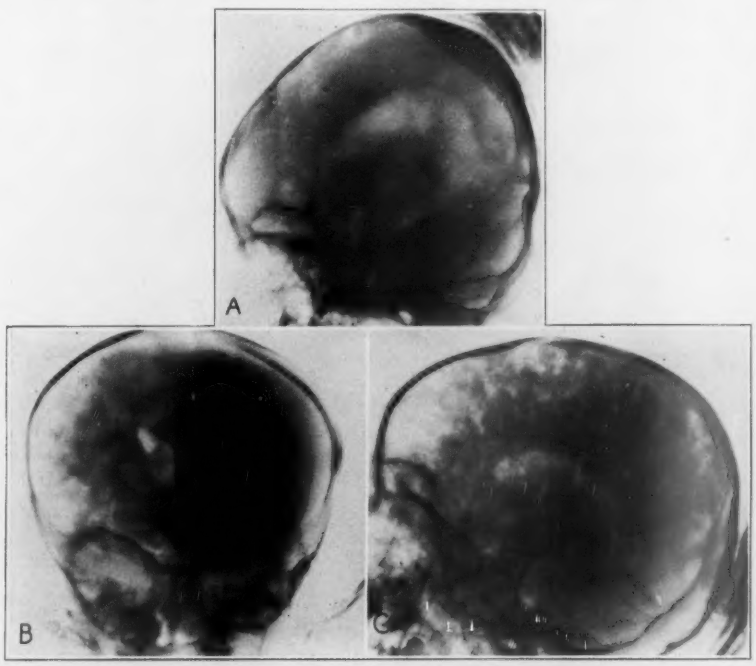

Fig. 6 (case 1).—*A*, cerebral maldevelopment. Malformation of head and ventricles. *B* (case 2), cerebral maldevelopment. Unilateral cortical atrophy with exaggerated cortical markings on right side; *C* (case 2), cerebral maldevelopment. Cortical atrophy and exaggerated cortical markings in frontal region.

Case 2.—W. R., a white boy, aged 1 year, was admitted to the Jewish Hospital because of inability to hold up his head, generalized weakness and marked irritability. He was born at term, was spontaneously delivered and weighed 10 pounds (4.5 Kg.) at birth. He was cyanotic at birth and did not cry until two days afterward. He presented no difficulty until 1 year of age, when he refused his feedings.

The patient was small and cried lustily. He presented a marked asymmetry of the head, the right side being much more prominent than the left. The anterior fontanel measured 3 by 3 cm. The frontal bosses were very prominent. There

was a continual rotatory nystagmus. Opacities were visible in both lenses. He followed light but not objects. There was marked beading at the costochondral junctions and flaring of the lower ribs. The spleen and liver were each palpable one fingerbreadth below the costal margin. There was marked hypotonicity of the muscles. He could not sit up or hold up his head. The clinical diagnosis was cerebral maldevelopment, bilateral congenital cataract and rickets.

Encephalography.—On the patient's fourth day in the hospital, 100 cc. of fluid was removed and 90 cc. of air injected. The cerebrospinal fluid was entirely normal on examination. The child became somewhat cyanotic, and respirations were shallow following the procedure, so that stimulation was resorted to. His temperature remained elevated for two days (from 103 to 104 F.) and then fell to normal. The encephalograms (figs. 6 *B* and *C*) showed normal configuration of the ventricles with excessive amounts of air in the subarachnoid space on the right side and practically none on the left. The cortical markings were exaggerated over the frontal and temporal regions in the lateral view.

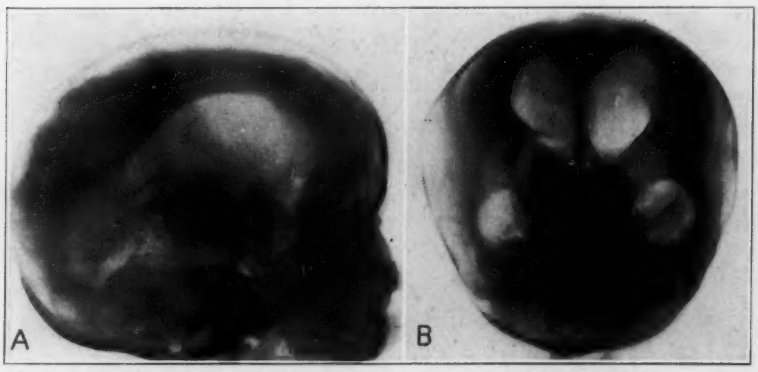

Fig. 7 (case 3).—*A*, tuberculous meningitis. Markedly dilated ventricles, lateral view. *B*, tuberculous meningitis, occipitofrontal view.

This case was probably one of cerebral cortical hemiatrophy.

Case 3.—V. C., a white girl, aged 7 months, was admitted to the hospital because of fever of one week's duration and cough of one day's duration.

The patient lay quietly in bed, cried occasionally and refused feedings. Tuberculin, 0.1 mg. injected intradermally, gave positive results. Roentgen examination revealed infiltration of the right upper lobe. Lumbar puncture yielded fluid under increased pressure, showing 100 lymphocytes per cubic millimeter; sugar was reduced and the Pandy test was positive. A pellicle formed on standing, but no tubercle bacilli could be demonstrated. The clinical diagnosis was pulmonary tuberculosis and tuberculous meningitis.

Encephalography.—On the second day in the hospital, 70 cc. of fluid was removed and 55 cc. of air injected. Except for a slight rise in temperature, there was no postoperative reaction. The encephalograms (figs. 7 *A* and *B*) showed generalized dilatation of the lateral ventricles. The third and fourth ventricles were not visualized. The left ventricle appeared larger than the right, as noted in the fronto-occipital view (fig. 7 *B*).

This case illustrates the early onset of hydrocephalus in tuberculous meningitis.

CASE 4.—J. C., a white girl, aged 6 months, was admitted to the Jewish Hospital on June 29, 1929, because of a large head and failure to follow light. There was nothing unusual in the birth or neonatal history. Feeding progressed normally without difficulty. When the patient was 3 months of age, routine examination by a physician revealed a disparity between the sizes of the head and the chest, the former measuring 17¼ inches (43.8 cm.) and the latter, 15¾ inches (38.9 cm.). Since that time no undue enlargement of the head had been observed.

Fig. 8 (case 4).—Hydrocephalus with dilatation of posterior horn of lateral ventricle, suggesting disease of the occipital lobe.

The patient was small, but fairly well nourished, and showed a uniform enlargement of the head. The anterior fontanel measured 6 by 7 cm.; the posterior fontanel and sutures were closed. The forehead was overhanging, and the eyes bulged. There was a slight horizontal nystagmus; the pupils reacted sluggishly to light, and examination of the fundi showed a definite pallor of the temporal half of each disk. The child did not follow light. The extremities were spastic and there was a bilateral spontaneous Babinski sign with no other abnormal neurologic findings. The clinical diagnosis was hydrocephalus.

Encephalography.—Two days after the patient's admission to the hospital, 70 cc. of fluid was removed and found to be normal; 65 cc. of air was injected. The temperature was elevated to 101 F. for two days following the procedure, and the child vomited once. The pulse was slow and irregular, and stimulation was given. The encephalogram (fig. 8) showed generalized dilatation of all ventricles, most marked, however, in the posterior horns of the lateral ventricles.

Five days after encephalography, the patient vomited and the temperature rose to 102 F. She was irritable and cried a great deal. The fontanel was found to be bulging. Lumbar puncture yielded cloudy fluid under moderately increased pressure with 8,000 cells per cubic millimeter, 89 per cent of which were lymphocytes. The sugar content was normal, and the globulin was slightly increased; no organisms could be demonstrated on smear or culture. Daily lumbar puncture for four days showed a gradual return of the fluid to normal, coincident with a decrease in temperature. It is noteworthy that one year after encephalography the child was still unable to see, which coincides with the picture of diseases of the occipital lobe, as shown by the excessively dilated posterior horns.

CASE 5.—D. G., a white girl, aged 1 month, was admitted to the hospital on May 22, 1929, because of a convulsion four days before admission and fever persisting since that time. She was a normal breast-fed infant until the onset of the present illness, when she became fretful and restless, suddenly stiffened out, rolled her eyes about, became cyanotic and vomited in a projectile manner.

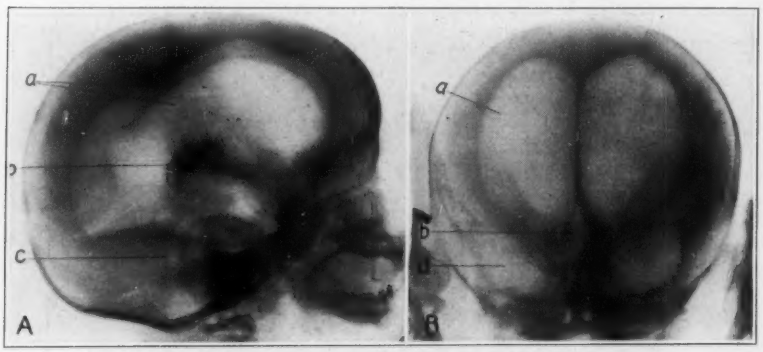

Fig. 9 (case 5).—*A*, cerebrospinal meningitis with dilatation of all ventricles. *B*, cerebrospinal meningitis and hydrocephalus, occipitofrontal view.

Physical examination revealed an irritable child with a bulging fontanel. No rigidity of the neck was demonstrable, and only a questionable Kernig sign could be elicited on the left side. Lumbar puncture yielded 25 cc. of cloudy fluid under increased pressure showing 6,400 cells, 94 per cent of which were polymorphonuclear leukocytes. A gram-negative diplococcus was present on direct smear, and meningococci on culture. Antimeningococcus serum was administered. When fluid could no longer be removed by the lumbar route, ventricular punctures were performed and serum was given thereby. Neutral phenolsulphonphthalein was injected into the ventricles and could not be recovered from the lumbar spinal canal. The clinical diagnosis was cerebrospinal meningitis and a block in the cerebrospinal fluid pathway.

Encephalography.—This was performed on June 17. The first 5 cc. of air to enter the spinal canal met with resistance; the subsequent 25 cc. entered much more readily. A roentgenogram failed to visualize the ventricles adequately. On June 24, air was injected into the left ventricle through the open fontanel; 150 cc. of normal fluid was withdrawn and replaced by an equal volume of air. The encephalograms (figs. 9 *A* and *B*) showed a marked generalized dilatation of all the ventricles.

The patient has been followed until the present time. She shows apparently normal development in that she can sit up and stand without support and can see and hear well. There is a slight weakness of the left foot. The head, though still 2 cm. larger than the chest, is growing at a less rapid rate than the latter.

This case illustrates an arrested hydrocephalus with subsequent apparently normal development.

CASE 6.—A. T., a white girl, aged 8 years, was admitted to the hospital on Nov. 9, 1929, because of headache of two months' duration, twitching of the left arm and failure of vision in the left eye of six weeks' duration. The patient had

Fig. 10 (case 6).—Abscess of the brain in the right precentral region. Note deformity of the right lateral ventrical and displacement of the entire ventricular system to the left.

chondrodystrophia and had been born with accessory fingers on each hand. One year previous to admission she had had purulent otitis media on the right side.

Physical examination showed a left internal strabismus, engorgement of the veins of the left retina, a slight facial weakness on the left side with flattening of the nasolabial fold, and a slight ataxia on the left side. The clinical diagnosis was abscess or tumor of the brain and chondrodystrophia.

Encephalography.—On May 18, 110 cc. of fluid was removed and proved to be entirely normal; 100 cc. of air was injected. The patient had headache and a temperature of 101 F. for one day, and vomited once after the procedure. The headache responded to acetphenetidin. The encephalogram (fig. 10) showed displacement of the entire ventricular system to the left and flattening of the upper part of the right lateral ventricle.

The patient was subsequently operated on by Dr. W. E. Dandy of Baltimore, and an abscess of the right precentral area was drained. The child recovered.

CASE 7.—A. T., a white girl, aged 3 months, was admitted on Nov. 14, 1929, because of convulsions and loss of weight. She was delivered spontaneously, but was cyanotic and required stimulation before respiration commenced. The infant did not take her feedings well and gained weight poorly. For fifteen days before admission she had been having convulsions of the right side of the face and body.

Physical examination revealed an apathetic child who breathed irregularly with marked periods of apnea. The right eyelids were held open wider than the left; the right pupil was larger than the left and both were fixed. There was a right-sided internal strabismus. Both fundi revealed a large number of disseminated atrophic areas in the choroid and retina. The deep reflexes were exaggerated. The clinical diagnosis was cerebral maldevelopment and convulsions.

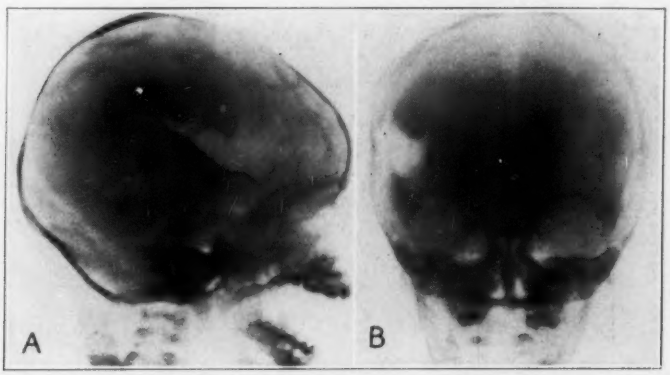

Fig. 11 (case 7).—*A*, cerebral maldevelopment showing areas of cortical atrophy. Ventricles not visualized. Lateral view. *B*, cerebral maldevelopment. Cortical atrophy. Ventricles not visualized. Fronto-occipital view.

Encephalography.—On November 23, 25 cc. of fluid was removed by the lumbar route and 20 cc. of air injected. This was insufficient to visualize the ventricles properly. Three days later, encephalography by the cisternal route was done, 45 cc. of fluid being withdrawn and 40 cc. of air injected. There was only a mild rise in temperature for two days. The encephalograms (figs. 11 *A* and *B*) showed a large irregular air shadow in what appeared to be the cortical area on the right side, indicating a porencephalic defect in the brain tissue. The ventricles could not be visualized.

SUMMARY

The accumulated experience of ten years with encephalography has served to evaluate its clinical significance. Our observations on twenty cases of encephalography in children are presented. Although encephalography is not without its contraindications and dangers, the procedure has proved to be of definite value in the differential diagnosis of many intracranial conditions and the exact localization of some of these. In

this way it has been helpful to surgeons contemplating operative intervention. In that it can reveal gross anatomic lesions, it is of distinct value to pediatricians and neurologists, especially in their consideration of prognosis. Therapeutic applicability has been demonstrated in some neurologic disorders.

MECKEL'S DIVERTICULUM

A PATHOLOGIC STUDY OF SIXTY-THREE CASES *

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Although in 1707 Ruysch [1] gave an excellent illustration of a diverticulum of the terminal ileum (fig. 1), it was Lavater,[2] in 1671, who first observed a case. In 1769, Morgagni [3] discussed diverticula of the duodenum, ileum and rectum; he did not believe, however, that those of the ileum had any relation to the vitelline duct.

In 1809,[4] his treatises contained not only a review of the literature of the sphenopalatine ganglion is still classic, that we are indebted for the clarity with which this developmental anomaly is now understood. In 1809,[4] his treatises contained, not only a review of the literature but a carefully worked out description of the development and anatomy of the diverticula that persist in the terminal ileum. He also brought out the relation of this process to the causation of disease.

EMBRYOLOGIC DATA [5]

It is normal for the proximal end of the yolk stalk to become obliterated early in fetal life. When it remains as a blind sac of varying lengths and attachment, it is known as Meckel's diverticulum.

The yolk sac performs the important function of the formation of blood during the first weeks of embryonal life, as the first blood cells and blood vessels develop in its outer mesodermal layer. In postfetal life, it remains only as a remnant.

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* Submitted for publication, Jan. 27, 1931.

* From the Pathological Laboratory of the Babies Hospital and the Department of Diseases of Children, College of Physicians and Surgeons, Columbia University.

1. Ruysch, F.: Thesaurus anatomicus, Amsterdam, J. Wolters, 1707, vol. 7, fig. 283.

2. Lavater, quoted by Kern, T.: Ueber die Divertikel des Darmkanals, Inaug. Diss., Tubingen, 1874 (from Morgagni).

3. Morgagni, G. B.: The Seats and Causes of Disease Investigated by Anatomy, Trans. from the Latin by Benjamin Alexander, London, A. Miller & T. Cadell, 1769, vol. 2, p. 141.

4. Meckel, J. F.: Ruil und Autenrieths, Arch. f. ges. Physiol. **9**:428, 1809; quoted by Fitz, H. R.: Persistent Omphalo-Mesenteric Remains; Their Importance in the Causation of Intestinal Duplication, Cyst Formation and Obstruction, Am. J. M. Sc. **88**:30, 1884; Handbuch der pathologischen Anatomie des Menschen-Auges, Hamburg, 1812, vol. 1, p. 553.

5. Keith, Arthur: Human Embryology and Morphology, London, E. Arnold, 1923.

In the second week of embryonic life, the yolk sac exists as a globular membrane that is attached to the intestine by a large opening (fig. 2). As the placenta develops, the yolk sac becomes pinched off and included in the substance of the placenta, and careful research will reveal its presence there at term. The only remaining connection between the yolk sac and the intestine is the yolk stalk, namely, the omphalomesenteric duct, which, with the development of the umbilical cord and its circulation, becomes merely a group of cells at the sixth week (fig. 3), traversing the entire length of the cord.

When the part of the yolk stalk that is connected with the intestine fails to obliterate itself at the intestinal wall, a pouch of varying length

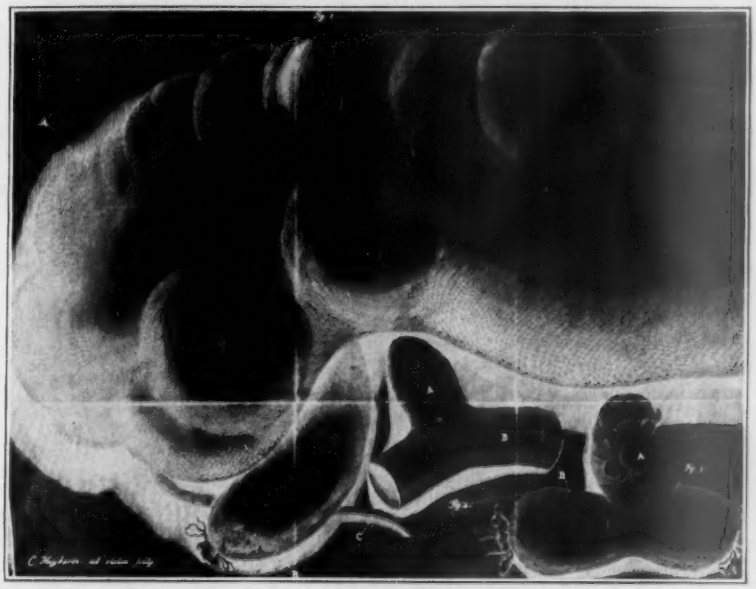

Fig. 1.—Photostatic copy of first illustration of Meckel's diverticulum. (From Ruysch: Thesaurus anatomicus, vol. 7, fig. 283.)

and attachments persists. This background will aid in the better understanding of the various types, complications and variations that are discussed in the following paragraphs.

INCIDENCE

The basis for this pathologic study is 63 cases of Meckel's diverticulum. This figure represents an incidence of 1.1 per cent in 5,768 consecutive necropsies performed on children at the Babies Hospital in the laboratory of Dr. Martha Wollstein.

In more than 10,000 laparotomies performed at the Mayo Clinic,[6] 15 cases of Meckel's diverticulum, an incidence of 0.14 per cent, were reported. At the other extreme is Adami,[7] who reported an incidence of 2.7 per cent, and Fuchsig,[8] who reported an incidence of 2 per cent. Telling[9] placed the incidence at 0.3 per cent in 13,068 necropsies. An average of these figures places the general incidence of Meckel's diverticulum at approximately 1 per cent.

SEX DISTRIBUTION

Ketteler[10] reported 109 cases of Meckel's diverticulum, of which 83.5 per cent occurred in males and 16.5 per cent in females. In a series of 1,635 coroners' cases, Mitchell[11] reported 39 cases, an inci-

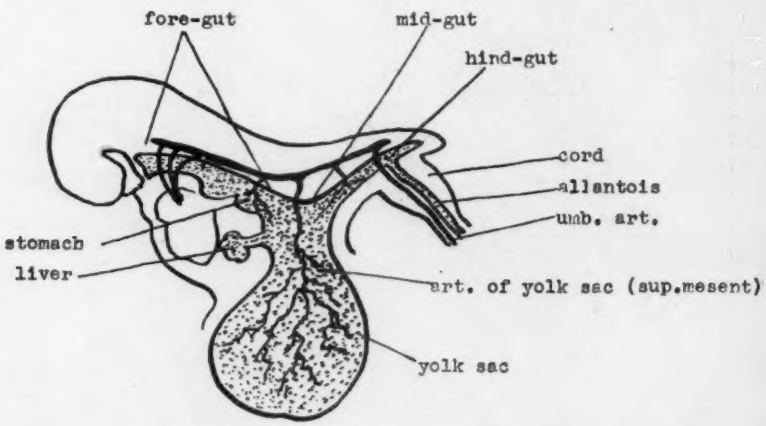

Fig. 2.—Form of the alimentary canal in a human embryo at the end of the fourth week.

dence of 2.4 per cent, of which 35 were in males and 4 in females. In my series of 63 cases, 74.6 per cent occurred in males and 25.4 per cent in females, a significant difference. This difference has long been observed, but a satisfactory explanation has not been offered.

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6. Balfour, D. C.: Meckel's Diverticulum: A Report of Fifteen Cases, J. Minnesota M. A. **31**:110, 1911.

7. Adami, J. G., and McCrae, J.: A Textbook of Pathology, Philadelphia, Lea & Febiger, 1912, p. 544.

8. Fuchsig, E.: Wien. klin. Wchnschr. **36**:730, 1923.

9. Telling, W. H. M.: Lancet **1**:843, 1908.

10. Ketteler, A.: Das Diverticulum meckelii als Ursache des Ileus, Göttingen, W. F. Kaestner, 1900.

11. Mitchell: J. Anat. & Physiol. **32**:675, 1897.

At the period of obliteration or nonobliteration of the omphalomesenteric duct, the genital ridge is beginning to become differentiated in the vicinity. This fact may be significant, though extensive search has failed to reveal a satisfactory explanation of the difference.

DISTANCE FROM ILEOCECAL VALVE

Great variation is possible in the site of Meckel's diverticulum. Fitz [12] expressed the belief that in the new-born child the distance from the ileocecal valve is 12 inches (30.48 cm.), and in the adult, it is 3 feet (91.44 cm.). Von Rokitansky [13] expressed the belief that it was from 1 to 2 feet (30.48 to 60.96 cm.) from the cecum. Foerster [14]

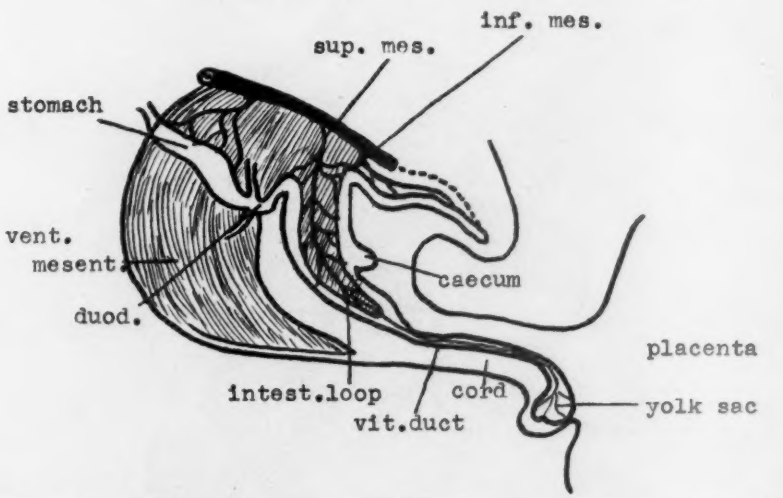

Fig. 3.—Form of the alimentary canal, the mesenteries and the arteries at the end of the sixth week.

gave a wider range, the upper limit of which was 4 feet (121.92 cm.), while Gray [15] gave 1 meter as the site in adults.

In this series, the location of the diverticulum in relation to the ileocecal valve also varied, 90 cm. (36 inches) being the upper limit, and 15 cm. (6 inches) the lower limit; the average was 50.6 cm. (20⅛ inches) in children under the age of 88 weeks.

12. Fitz (footnote 4).

13. von Rokitansky, C.: Lehrbuch der pathologischen Anatomie, ed. 3, Vienna, 1861, p. 182.

14. Foerster, A.: Handbuch der pathologischen Anatomie, ed. 2, Leipzig, 1863, vol. 2, p. 97.

15. Gray, H.: Anatomy of the Human Body, ed. 21, Philadelphia, Lea & Febiger, p. 1182.

TYPES OF MECKEL'S DIVERTICULA

With a review of the embryology in mind, it is possible to visualize any site and any variation in the size and shape of Meckel's diverticula. However, there are three main types and several subtypes: 1. Those that are free in the peritoneal cavity. In this group, representing 82.5 per cent, the diverticula are of varying lengths and dimensions; some have broad bases (fig. 4) with a lumen as large as the intestine itself; others have narrow bases with a diverticulum from 7 to 8 cm. in length (fig. 5). Von Rokitansky reported one 10 inches (25.4 cm.) in length, and Moll [16] reported a giant diverticulum 33½ inches (85.09

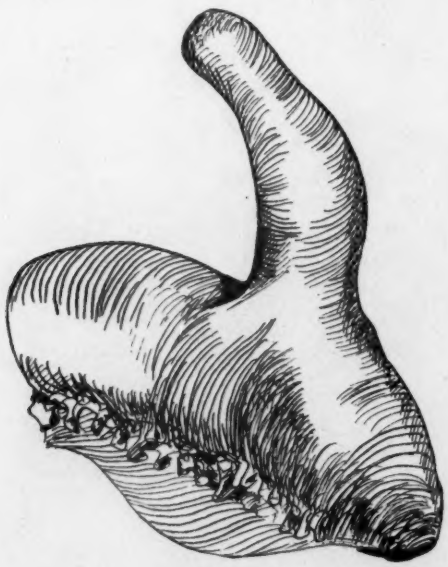

Fig. 4.—Meckel's diverticulum lying free in the abdominal cavity.

cm. long). One of those that were free in the abdomen showed a rare condition, bifurcation of the tip. Hyrtl [17] stated that this condition is extremely rare, and he presented a case in which the diverticulum was not only bifurcated, but serrated into five distinct parts. One specimen in my collection also showed multiple serration (fig. 6).

In another subgroup of the diverticula that are free in the peritoneal cavity there are mesenteries (fig. 7). One case in my series is represented in figure 7. Another diverticulum was bound firmly to the intestine and followed its general course for a distance of 3 cm.

16. Moll, H. H.: Giant Meckel's Diverticulum, Brit. J. Surg. **14**:176, 1926.

17. Hyrtl, J.: Handbuch der topographischen Anatomie, Vienna, W. Braumüller, 1860, vol. 1, p. 642.

Fig. 5.—Meckel's diverticulum with a broad base.

Fig. 6.—Meckel's diverticulum, showing bifurcation of the tip.

(fig. 8). In one case, a thickened tip of Meckel's diverticulum revealed typical gastric glands in the mucosa. This has been reported by numerous authors and has occurred in as many as 16.6 per cent of 30 cases examined by Schaetz.[18] Guibal[19] expressed the belief that this type of Meckel's diverticulum ulcerates, causes hemorrhage and occasionally perforates with consequent peritonitis. He agreed with Stulz and Wörringer[20] that the cause of hemorrhage or perforation in Meckel's diverticulum is "to be found in the ulceration of heteroplastic gastric mucosa." In my case, the lumen of the diverticulum contained blood, but ulceration was not present. There were 5 cases in which perforation, ulceration or erosion was present without evidence of gastric mucosa in microscopic sections.

2. Those in which the patency of the omphalomesenteric duct persists after birth, and in which there is direct communication between

Fig. 7.—Meckel's diverticulum with a broad mesentery.

the outside and the intestine. A fecal fistula results. In this group herniations of the entire peritoneal contents also occur; 4 cases of the series were of this type.

3. Those in which the omphalomesenteric duct remains patent at its proximal (intestinal) insertion and is attached to the umbilicus by a fibrous cord, as a result of which serious complications often result. This group represents 6 or 10 per cent of my series. Externally, an umbilical polyp is usually noted, although this sign was not observed in my series (Cullen[21]).

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18. Schaetz, G.: Beitr. z. path. Anat. u. z. allg. Path. **74**:115, 1925.

19. Guibal, L.: Peptic Ulcer of Meckel's Diverticulum Causing Profuse Intestinal Hemorrhage, Bull. et mém. Soc. nat. de chir. **50**:349, 1924.

20. Stulz, E., and Wörringer, P.: Peptic Ulcer in Meckel's Diverticulum in Children, Paris méd. **57**:387 (Nov.) 1925; abstr., J. A. M. A. **85**:1921 (Dec. 12) 1925.

When such abnormal peritoneal bands exist, the danger of surgical complications is increased many fold. Volvuli and intestinal obstruction often result in this type of diverticulum, as will be discussed later.

Occasionally, one does not find a true Meckel's diverticulum but merely its remnant, which is represented by a fibrous cord containing the degenerated omphalomesenteric vessels and duct. This condition may also be associated with serious complications.

COMPLICATIONS

Complications occurred in 10 cases, or 15.8 per cent of this series of 63 cases.

Five, or 50 per cent, of the complications in this series were associated with umbilical herniation. In 1 case there was an intestinal

Fig. 8.—Meckel's diverticulum following the course of, and bound to, the ileum.

obstruction, but this condition was associated with abnormal peritoneal bands. In 8 of the 10 complicated cases, there was evidence of inflammation in the region of Meckel's diverticulum with general peritonitis in 5. In 8 of the 10 cases with complications, the patients were males. Wellington,[22] reviewing a series of 326 cases of Meckel's diverticulum that were associated with pathologic conditions, found that in 201 (61.6 per cent) the patients were males; in 64 (19.6 per cent) the patients were females, and in 61 cases the sex was not given.

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21. Cullen, T. S.: Embryology, Anatomy and Diseases of the Umbilicus, Philadelphia, W. B. Saunders Company, 1916.

22. Wellington: Surg., Gynec. & Obst. **16**:74, 1913.

MALFORMATIONS ASSOCIATED WITH MECKEL'S DIVERTICULUM

Rather extensive search of the literature has failed to reveal a reference concerning the coincidence of malformations with Meckel's diverticulum, although the association has long been well known. In the series reported, there were multiple malformations and in 13 cases,

TABLE 1.—*Complications in Cases of Meckel's Diverticulum*

Case	Sex	Age in Weeks	Complications
1	M	1	Umbilical herniation; perforation with peritonitis
2	M	30	Umbilical herniation; perforation, abnormal peritoneal bands
3	M	3	Erosions of diverticulum
4	M	44	Umbilical herniation; perforation with peritonitis
5	M	17	Umbilical herniation; perforation with peritonitis
6	M	1	Omphalitis; peritonitis, abnormal peritoneal bands
7	F	1	Omphalitis; abnormal peritoneal band
8	M	74	Abnormal peritoneal bands with intestinal obstruction
9	F	1	Umbilical herniation with perforation
10	M	2	Ulceration with peritonitis

TABLE 2.—*Malformations Associated with Meckel's Diverticulum*

Case	Sex	Associated Anomaly
1	M	Congenital cardiac malformation; microgyria; harelip; cleft palate; accessory spleen; supernumerary lobe in the lungs
2	M	Microcephaly
3	M	Congenital hypertrophic pyloric stenosis
4	M	Spina bifida
5	F	Mongolism; congenital cardiac malformation
6	F	Supernumerary spleen
7	M	Congenital cardiac malformation
8	M	Stenosis of the larynx
9	M	Mesenteric cyst
10	M	Clubfeet
11	M	Supernumerary pancreas; atresia of the esophagus
12	F	Malformation of the gallbladder; bilateral malformation of the ureters
13	M	Supernumerary spleen; horseshoe-kidneys; congenital abnormal peritoneal bands
14	M	Congenital cardiac malformation; cleft ureter
15	F	Malformation of the brain; supernumerary spleen; malformation of the gall-bladder; hydronephrosis
16	M	Horseshoe-kidney
17	M	Congenital cardiac malformation
18	M	Congenital hypertrophic pyloric stenosis
19	M	Mongolism
20	M	Supernumerary spleen
21	M	Congenital cardiac malformation

single malformations, which gives a total of 33 per cent of the patients with malformations associated with Meckel's diverticulum.

This observation is undoubtedly of clinical significance, and the presence of congenital anomalies will serve to remind one to search for this abnormality of the gastro-intestinal tract. It is interesting to note that 80.9 per cent of the cases of associated anomalies also occurred in males.

Perhaps the large percentage of associated anomalies suggests a primary defect in the germ plasm as a possible etiologic factor.

SUMMARY AND CONCLUSIONS

1. A short history of the literature on Meckel's diverticulum is presented, and a series of 63 such cases is reported from the pathologic standpoint.

2. The embryology is discussed as a basis for a better understanding of the anatomy and pathology of the diverticuli of the terminal ileum.

TABLE 3.—*Associated Anomalies Grouped as to Systems*

System	Number of Cases
Brain and spinal cord	7
Congenital cardiac malformations	6
Kidneys and genito-urinary system	5
Spleen	5
Mongolism	3
Stomach (pyloric stenosis)	2
Atresia of the esophagus	1
Pancreas	1
Gallbladder	1
Mesenteric cyst	1
Congenital abnormal peritoneal bands	1
Stenosis of the larynx	1
Lung	1
Harelip and cleft palate	1
Extremities	1

3. Comparisons with previous groups substantiate observation of a preponderance of males (74.6 per cent), and set the probable incidence of Meckel's diverticulum at 1 per cent in a large series (5,768) of necropsies.

4. The site of Meckel's diverticulum in relation to the ileocecal valve varies within wide limits.

5. Types of Meckel's diverticulum are discussed and illustrated.

6. The complications are listed, and the coincidence of other anomalies associated with Meckel's diverticuli in 33 per cent of the cases is offered as evidence of a defective germ plasm as an etiologic factor.

ELECTROCARDIOGRAPHIC STUDIES IN INFECTIOUS DISEASES

II. SCARLET FEVER *

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AND
LEO M. TARAN, M.D.
BROOKLYN

There are many points of similarity between scarlet fever and rheumatic fever.

The streptococcus plays an important rôle in both diseases. G. F. Dick and G. H. Dick [1] showed that the streptococcus is the specific organism in scarlet fever. Since 1900, when Poynton and Paine [2] described the diplostreptococcus as the specific organism in rheumatic fever, many workers (Rosenow,[3] Faber,[4] Swift and Kinsella [5] and Small [6]) have described one or another strain of the streptococcus group as an etiologic agent in that disease.

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* Submitted for publication, Jan. 21, 1931.

* From the Cardiological Departments of the Jewish Hospital of Brooklyn and the Kingston Avenue Hospital for Contagious Diseases.

* Read before the Meeting of the Clinical Society of the Kingston Avenue Hospital on Oct. 9, 1930.

1. Dick, G. F., and Dick, G. H.: Experimental Inoculations in Scarlet Fever, J. A. M. A. **77**:782 (Sept. 3) 1921; Experimental Scarlet Fever, ibid. **81**:1166 (Oct. 6) 1923; Etiology of Scarlet Fever, ibid. **82**:301 (Jan. 26) 1924.

2. Poynton, F. J., and Paine, A.: Etiology of Rheumatic Fever, Lancet **2**:860 (Sept. 22), 932 (Sept. 29) 1900.

3. Rosenow: The Etiology of Acute Rheumatism, Articular and Muscular, J. Infect. Dis. **14**:61, 1914.

4. Faber, H. K.: Experimental Arthritis in the Rabbit; a Contribution to the Pathogeny of Arthritis in Rheumatic Fever, J. Exper. Med. **22**:615, 1915.

5. Swift, H. F., and Kinsella, R. A.: Bacteriologic Studies in Rheumatic Fever, Arch. Int. Med. **19**:381 (March) 1917.

6. Small, J. C.: Streptococci in Relation to Rheumatic Disease, M. Clin. North America **13**:857 (Jan.) 1930; Bacterium Causing Rheumatic Fever and Preliminary Account of Therapeutic Action of Its Specific Antiserum, Am. J. M. Sc. **173**:101 (Jan.) 1927; Rheumatic Fever; Observations Bearing on Specificity of Streptococcus Cardioarthritidis in Rheumatic Fever and Sydenham's Chorea, ibid. **175**:638 (May) 1928.

Escherich and Schick [7] and Swift [8] suggested that many of the symptoms occurring in scarlet fever—adenopathy, recurrent angina, arthritis and simple endocarditis—are allergic in nature. Experimental studies by Swift and his co-workers,[9] Mackie and McLachlan [10] and Zinsser and Grinnel [11] seem to point to the fact that the allergic phenomena in scarlet fever are an expression of hypersensitivity to *Streptococcus hemolyticus.*

Swift,[12] Bezançon and Weil,[13] Birkhaug [14] and others brought forth more than plausible evidence to show that at least many of the symptoms of rheumatic fever can be explained by the existence of a state of hypersensitivity to streptococci. Experimentally, Swift [15] and his co-workers, Mackenzie and Hanger [16] and others showed the lack of strain specificity of the various groups of streptococci both in persons with rheumatic fever and in animals. The stimulus to the development of the hypersensitive state in that disease may, therefore, be any one of the various strains of the streptococcus group that has been obtained from the blood of patients with rheumatic fever.

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7. Escherich and Schick: Scharlach, Vienna, Hölder Pichler-Tempsky A.-G., 1912.

8. Swift, H. F.: The Heart in Infection, Am. Heart J. **3**:632 (Aug.) 1928.

9. Andrews, C. H.; Derick, C. L., and Swift, H. F.: Hemolytic Streptococci in Acute Rheumatic Fever, with Analysis of Antigenic Relationship Existing Among Certain Strains, J. Exper. Med. **43**:13 (Jan.) 1926. Derick, C. L., and Swift, H. F.: Reactions of Rabbits to Nonhemolytic Streptococci; General Tuberculin-like Hypersensitiveness, Allergy or Hyperergy Following Secondary Reaction, ibid. **49**:615 (April) 1929. Hitchcock, C. H., and Swift, H. F.: Studies on Indifferent Streptococci; Allergizing Capacity of Different Strains, ibid. **49**:637 (April) 1929. Swift, H. F.: Rheumatic Fever, Am. J. M. Sc. **170**:631, 1925.

10. Mackie, T. J., and McLachlan, D. G. S.: Experimental Sensitization to Culture-Filtrates of Streptococcus Scarlatinae, Brit. J. Exper. Path. **8**:129, 1927.

11. Zinsser, H., and Grinnel, F. B.: Allergic Reactions to Hemolytic Streptococcus, J. Immunol. **10**:725, 1925.

12. Swift, H. F.; Derick, C. L., and Hitchcock, C. H.: Bacterial Allergy (Hyperergy) to Nonhemolytic Streptococci in its Relation to Rheumatic Fever, J. A. M. A. **90**:906 (March 24) 1928.

13. Bezançon, F., and Weil, M. P.: La spécifité de la malade rheumatismale, Ann. de méd. **19**:81, 1926.

14. Birkhaug, K. E.: Rheumatic Fever; Allergic Reactions with Toxin-Producing Strain of Nonmethemoglobin-Forming Streptococcus Isolated from Rheumatic Fever, J. Infect. Dis. **43**:280 (Oct.) 1928.

15. Swift, H. F.: The Heart in Infection, Am. Heart J. **3**:636 (Aug.) 1928.

16. Mackenzie, G. M., and Hanger, F. M.: Allergic Reactions to Streptococcus Antigens, J. Immunol. **13**:41, 1927.

Not infrequently a patient with a definite case of scarlet fever will present all the typical signs of rheumatic fever. The rekindling of rheumatic carditis in the course of scarlet fever is well known. Hector [17] emphasized the frequency and the severity of cardiac involvement in patients with scarlet fever who gave a history of previous rheumatic fever.

There are many controversial points in the pathology of these diseases. Aschoff [18] and also Geipel [19] described the submiliary nodule as specific in rheumatic fever. The pathologic process in scarlet fever is, however, less specific. Schmorl, cited by Fahr, described submiliary nodules somewhat similar to the Aschoff nodes in the myocardium of patients dying from scarlet fever. Fahr [20] stated that the perivascular reactions found in the cardiac muscle in fatal cases of this disease differ from the typical Aschoff body only in that they do not contain giant cells. He stated further that the myocardial lesions found in rheumatic carditis often cannot be distinguished from the lesions found in scarlatinal carditis. Coombs [21] expressed the belief that the lesions found in scarlatinal carditis are not simply identical with, but really are the lesions of, rheumatic carditis. Many symptoms—tonsillar involvement, manifestations in the joints, cardiac complications and nephritis—are common to both diseases.

These controversial observations have at least emphasized the similarity between scarlet fever and rheumatic fever. We thought, therefore, that a comparison of the electrocardiographic observations in scarlet fever with those reported in rheumatic fever would be of interest.

Fifty patients, admitted consecutively to the Kingston Avenue Hospital for Contagious Diseases, were studied. The diagnosis was made by the history of the onset, the rash, the characteristic tongue and the desquamation. No doubtful cases were included, and no children having a previous history of rheumatic fever were studied. Forty-one cases were mild, eight moderately severe, and only one was severe.

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17. Hector, F. J.: Effects of Scarlet Fever on Rheumatic Subjects, Arch. Dis. Childhood **1**:339, 1926.

18. Aschoff, L.: Zur Myocarditis Frage, Behandl. der deutsch. path. Gesellsch. **7**:46, 1924.

19. Geipel, P.: Untersuchungen über rheumatische Myocarditis, Deutsches Arch. f. klin. Med. **85**:75, 1925.

20. Fahr, T.: Beiträge zur Frage der Herz- und Gelenkveränderungen bei Gelenkrheumatismus und Scharlach, Virchows Arch. f. path. Anat. **232**:134, 1921.

21. Coombs, C. F.: Rheumatic Heart Disease, New York, William Wood & Company, 1924, p. 29 and p. 242.

The Cambridge portable electrocardiograph was used. The use of immersion electrodes being under the circumstances impracticable, a modification of a method described by Herrmann and Wilson [22] was used. Flannel bandages, one thickness, saturated in a warm saline solution, were applied to the extremities and over the bandages were wound malleable lead wires, ⅛ inch in thickness and 3 feet long, which were connected by a Faunstock clip to the wire leads from the machine. There was very little overshooting with this method.

Records were taken every other day in most cases, from the day of admission to the time of discharge. Scarlet fever antitoxin was not given in all of our cases; only 30 per cent of the patients in our series of milder cases received it; in the moderately severe cases, 50 per cent of the patients received antitoxin, and the patients with very severe cases were given large doses of antitoxin. No cardiac drugs of any kind were given during the course of this study. All tracings were taken at approximately the same hour daily.

RATE

In cases of scarlet fever studied in the afebrile stage, the rates did not differ materially from the rates in normal persons (table 1 and fig. 1). There were more children with rates over 100 in the febrile than in the afebrile periods. Twenty-seven per cent of the febrile cases showed a rate of from 100 to 110; 24 per cent from 110 to 120, and 24 per cent from 120 to 130. In the febrile periods the increase in rate seemed only commensurate with the increase in temperature (table 1 and fig. 2).

TABLE 1.—*Rate*

Rates	Normal	Scarlet Fever		
		Febrile Stage in Scarlet Fever, Percentage of Cases	Afebrile Stage in Scarlet Fever, Percentage of Cases	Total, Percentage of Cases
60–70	3.2	6.0	....	2.6
70–80	9.6	3.0	6.0	3.3
80–90	20.0	....	20.0	15.3
90–100	32.4	6.0	31.0	26.0
100–110	18.4	27.0	16.5	18.3
110–120	6.4	24.0	14.0	16.0
120–130	4.8	24.0	2.6	7.4
130–140	3.2	2.0	6.0	5.3
140–150	1.6	5.0	3.5	4.0
150–160	....	2.5	....	1.0

22. Herrmann, G. R., and Wilson, F. N.: A New Electrode for Clinical and Experimental Electrocardiography, Am. Heart J. **1**:111, 1925.

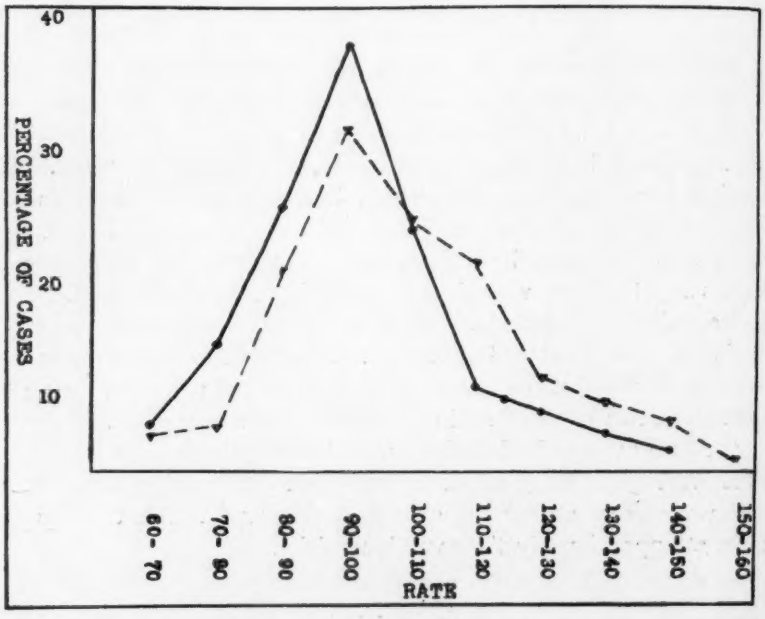

Fig. 1.—These curves illustrate the percentage of incidence of various rates in normal children and in those with scarlet fever. The ordinates represent the percentage of cases and the abscissas, the various rate levels. The solid line is the curve for normal children, while the dash line is the curve for patients with scarlet fever.

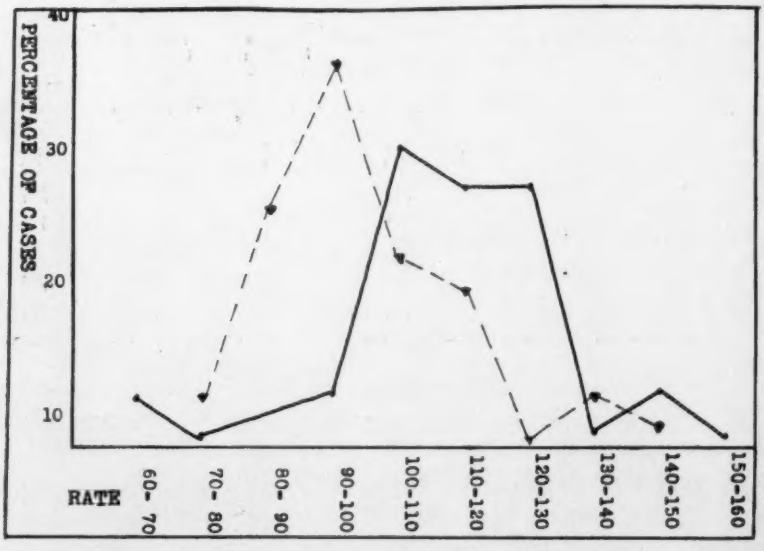

Fig. 2.—These curves are plotted to show a comparison in the rate incidence between the febrile and the afebrile cases of scarlet fever. The solid line represents the curve for the febrile cases and the dash line represents the afebrile cases.

ARRHYTHMIAS

Bradycardia.—A decided decrease in the cardiac rate occurred in the early part of the disease in approximately 25 per cent of the children. The average decrease was about 33 beats per minute. This relative bradycardia was first noticed as early as the fourth day and as late as the thirteenth day of the disease; as a rule, however, it occurred about the tenth day and returned to the normal rate within a period of three weeks from its onset (table 2, figs. 3, 4 and 5). A sinus arrhythmia was noticed in all these children. This slowing of the rate occurred in the febrile as well as the afebrile stages. In only seven of these twelve cases was antitoxin administered. In one of the children, in whom a definite slowing of the pulse rate was noticed on the tenth day of the

TABLE 2.—*Bradycardia*

Patient	Age, Years	Rate Before Brady-cardia	Day of Appear-ance	Tempera-ture, F.	Rate	Day of Dis-appear-ance	Tem-pera-ture, F.	Rate After Brady-cardia	Anti-toxin
F.W.....	6	111	12	98.6	76	20	Normal	108	Yes
S.H.....	5½	130	11	100.0	84	24	Normal	111	Yes
V.S.....	7	107	4	102.4	65	8	Normal	143	Yes
W.M.....	8	104	10	98.0	63	15	Normal	107	Yes
V.L.....	11	100	5	101.0	76	12	Normal	108	No
S.B.....	6	136	12	99.4	86	20	Normal	107	Yes
H.L.....	7	107	11	98.6	73	27	Normal	111	No
G.M.....	5	105	13	99.4	70	20	Normal	115	No
O.O.....	8	105	8	98.6	75	15	Normal	96	Yes
M.E.....	13	90	8	99.2	72	15	Normal	120	Yes
G.S.....	11	100	10	98.6	75	14	Normal	100	No
O.I.....	12	85	10	98.6	65	16	Normal	78	No

illness, tachycardia developed on the twelfth day without any apparent reason and with no rise in temperature; two days later, however, measles developed.

Swift [8] suggested that bradycardia in scarlet fever is an evidence of myocardial involvement. Only one of the children in whom bradycardia developed showed evidence of myocardial involvement by abnormalities in the electrocardiograph. All the children had a sinus arrhythmia. We believe, therefore, that the slow rate is an evidence of an extracardiac imbalance in the nervous mechanism rather than of myocardial involvement. This bradycardia is apparently similar to the post-infectious bradycardias except that in scarlet fever it occurs in the early part of the disease.

Sinus Arrhythmia.—The frequency of occurrence of sinus arrhythmia in scarlet fever seems to equal its occurrence in normal children in the same age periods. It is observed less frequently in the febrile stages, while in the afebrile period it parallels in frequency that in

normal children. It was present in all the children in whom bradycardia developed during the course of the disease (table 3).

Extrasystoles.—No extrasystoles were observed.

TABLE 3.—*Rhythm*

	Normal	Scarlet Fever		
		Febrile, Percentage of Cases	Afebrile, Percentage of Cases	Total, Percentage of Cases
Sinus Arrhythmia	78	62.5	85	78
Sinus Tachycardia	8	37.5	15	22
Regular Sinus Rhythm	14	....	..	..

Fig. 3.—Electrocardiographic tracings showing the bradycardia on the eighth day of the disease in a child aged 8 years. The tracings were made on the fifth day (upper row), eighth (second row), eleventh (third row) and fifteenth (fourth row) days. Note the changes in the direction of T^3.

P WAVE

In general, no abnormalities of the P wave were noted. In one case the direction of the P wave in the third lead was constantly changing. In another case the P^3 was found to be inverted on the ninth day (fig. 6) and upright on the following day. Such changes are seen in normal children and were not considered indicative of disease.

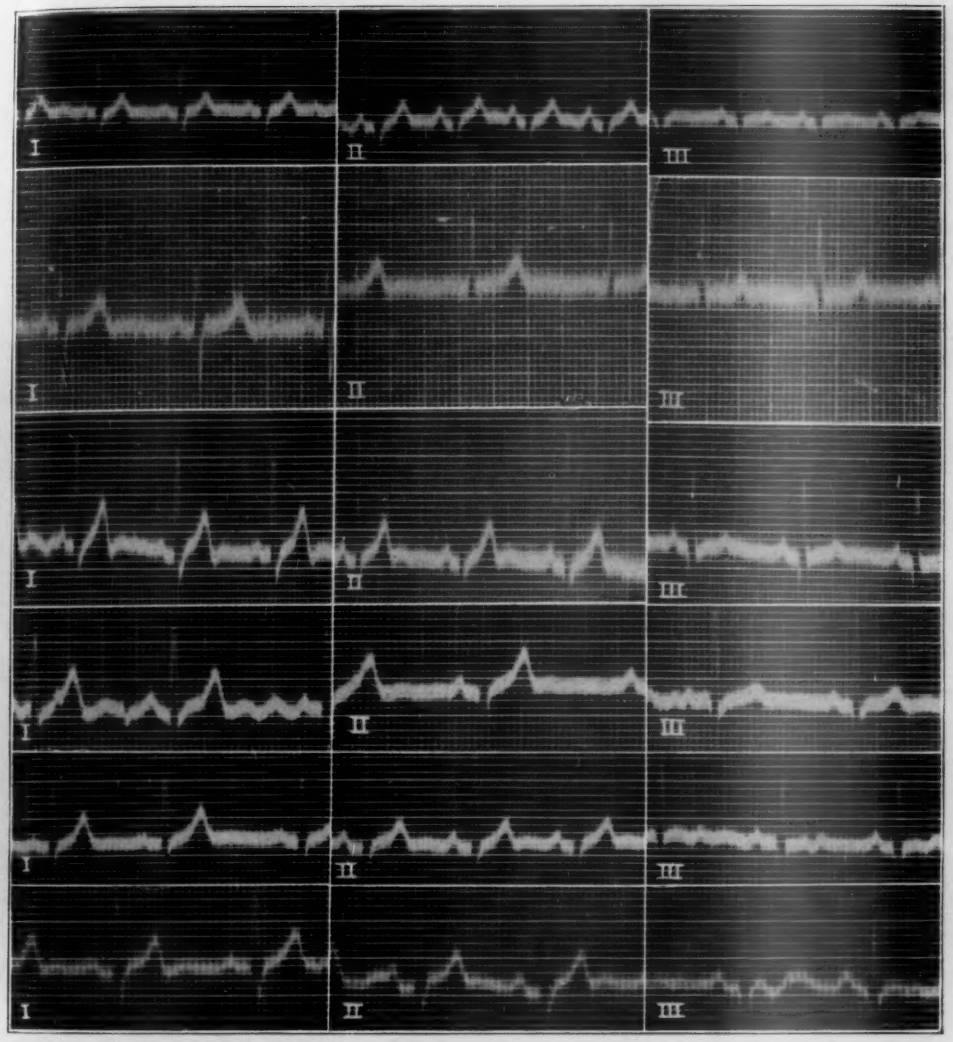

Fig. 4.—The tracings illustrate the occurrence of the right axial deviation on the sixth day of illness with a gradual return to a normal deviation of the axis on the tenth day and also shows a bradycardia on the thirteenth day. This bradycardia, which occurred in a child aged 12 years, began on the sixth day of the illness and was most marked on the thirteenth day. Note the changing direction of the T wave in the third lead. The tracings were taken on the fourth day (first row, temperature 100.6 F.), and on the sixth (second row, temperature 99.0 F.), the tenth (third row, temperature 99.0 F.), the thirteenth (fourth row, temperature 98.6 F.), the sixteenth (fifth row, temperature 98.6 F.) and the twenty-first day (sixth row, temperature 98.6 F.).

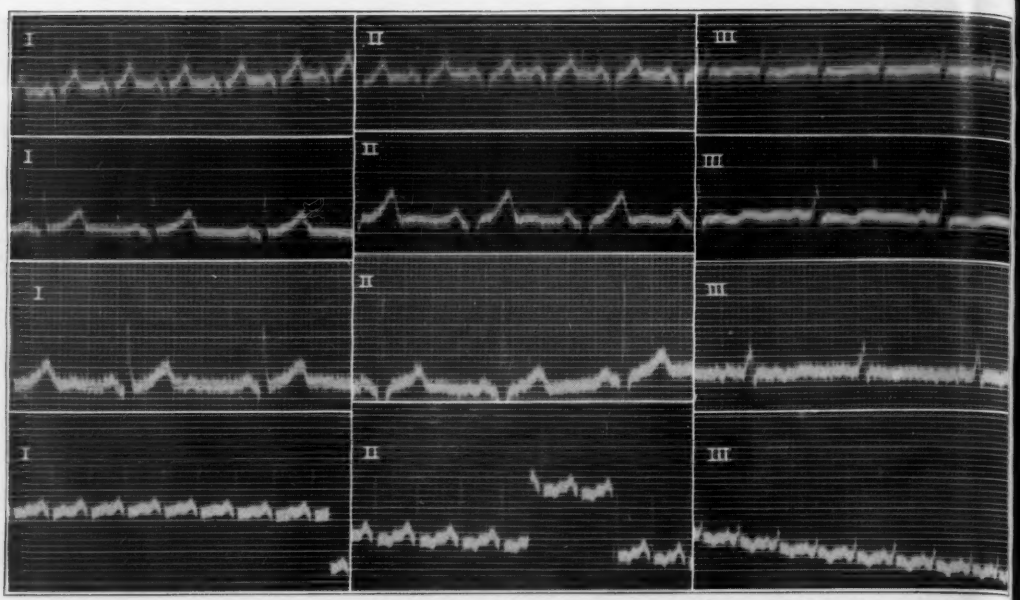

Fig. 5.—Bradycardia on the eighth day of the disease; most marked on the eleventh. Note the inverted T^1, T^2 and T^3 on the fifteenth day of the disease, also the poor R-T transition in lead I on the eighth day. These tracings were made on the seventh day (upper row), eighth (second row), eleventh (third row) and fifteenth (fourth row) days in the illness of a child aged 13 years.

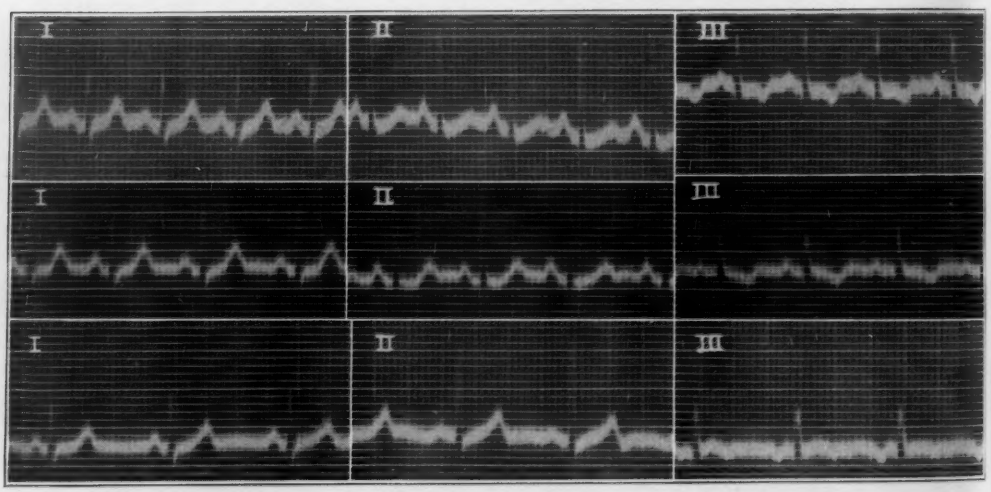

Fig. 6.—Tracings made on the third day (upper row, temperature 99 F.), sixth (second row, temperature 98.6 F.) and on the ninth (third row, temperature 98.8 F.) days of illness in a child aged 9 years. Note the R-T interval and the changes in the T wave in the second leads; also the inversion of P^3 on the ninth day.

The highest P waves were found in the febrile cases. The maximum height was 4 millivolts; the minimum height was 1.5 millivolts. The average height in the scarlet fever cases in the afebrile stage was 1.9 millivolts as compared with 1.7 millivolts in normal children (table 4).

TABLE 4.—*P Wave*

	Normal	Scarlet Fever		
		Average	Febrile	Afebrile
Highest	2.5 mv.	4.0 mv.	4.0 mv.	2.5 mv.
Lowest	1.3 mv.	1.5 mv.	1.5 mv.	1.5 mv.
Average	1.7 mv.	2.4 mv.	3.0 mv.	1.9 mv.

P-R INTERVAL

The P-R interval ranged from 0.08 to 0.18 second. In the patients with fever, the maximum P-R interval was 0.14 and the minimum 0.08 second. In the afebrile patients the maximum P-R interval was 0.18 and the minimum 0.08 second. There seems to be a tendency for this interval to be shorter in the febrile period of the disease. These figures do not differ from those observed in normal children, the average length for similar age groups in the normal subjects being 0.125 second (table 5).

TABLE 5.—*P-R Interval*

	Normal	Scarlet Fever		
		Average	Febrile	Afebrile
Maximum	0.17 sec.	0.18 sec.	0.14 sec.	0.18 sec.
Minimum	0.08 sec.	0.08 sec.	0.08 sec.	0.08 sec.
Average	0.125 sec.	0.125 sec.	0.115 sec.	0.125 sec.

The standard deviation of this interval in this series of cases was found to be 0.0208 second. As previously mentioned, the standard deviation for our normal series was 0.0220 second.

The P-R interval was measured in fifty cases of rheumatic fever in children between the ages of 6 and 14 years. The standard deviation in this series was found to be 0.0340 second. The average length of the P-R interval was 0.1936 second (table 6).

TABLE 6.—*Standard Deviation (P-R Interval)*

	Mean Deviation	Standard Deviation
Normal	0.1244 sec.	0.0220 sec.
Scarlet fever	0.1256 sec.	0.0208 sec.
Diphtheria	0.1274 sec.	0.0202 sec.
Rheumatic fever	0.1936 sec.	0.0340 sec.

QRS INTERVAL

The average QRS interval was 0.055 second; the average for the febrile cases was 0.042 second and for the afebrile cases, 0.063 second. This interval is shorter in the febrile periods of the disease (table 7).

No abnormalities in the shape of this complex were observed in the first or second leads. In the third lead, however, 50 per cent of the cases showed a slurring or a slight widening. This percentage is slightly higher than that in normal children.

TABLE 7.—*QRS Interval*

		Scarlet Fever		
	Normal	Average	Febrile	Afebrile
Maximum	0.06 sec.	0.12 sec.	0.08 sec.	0.12 sec.
Minimum	0.03 sec.	0.02 sec.	0.02 sec.	0.03 sec.
Average	0.046 sec.	0.055 sec.	0.042 sec.	0.063 sec.

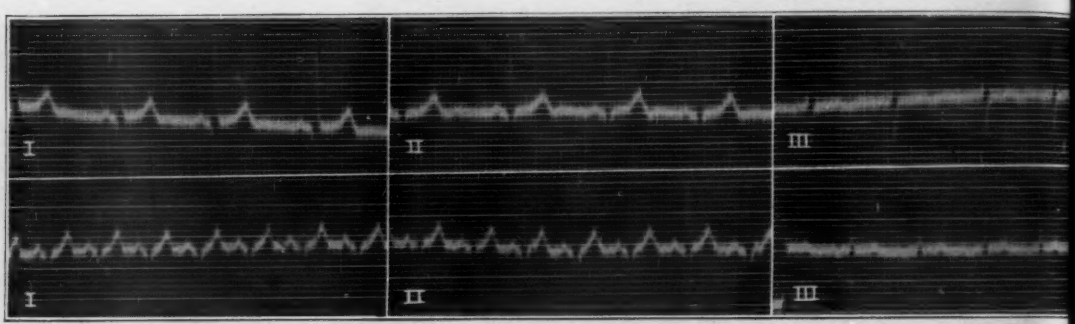

Fig. 7.—Tracings made on the fourth and seventh days (upper and lower row, respectively) of illness in a child aged 11 years. A slight abnormality was shown in the R-T transition on the fourth day of the disease. Note the left axial deviation which returned to normal on the seventh day.

R-T OR S-T INTERVAL

The S-T interval ranged from 0.02 to 0.22 seconds. The average for the febrile cases was 0.105 second, and for the afebrile 0.12 second (table 8).

TABLE 8.—*S-T Interval*

		Scarlet Fever		
	Normal	Average	Febrile	Afebrile
Maximum	0.16 sec.	0.22 sec.	0.14 sec.	0.22 sec.
Minimum	0.06 sec.	0.02 sec.	0.02 sec.	0.03 sec.
Average	0.115 sec.	0.115 sec.	0.105 sec.	0.12 sec.

In four children, or 8 per cent of those observed, disturbances in the position of this interval were observed. In two children the R-T^1 was above the base line and in two children it was below. In one of these cases there were concomitant T^1, T^2 and T^3 changes. These abnormalities occurred in the early days of the disease and disappeared before the patient was discharged from the hospital (figs. 6 and 7).

The infrequency of disturbances in the position of this interval in scarlet fever brings to mind the frequency of such disturbances in rheumatic fever. Rothschild, Sacks and Libman [23] described a definite distortion of this interval in rheumatic fever.

T WAVE

The height of the T wave did not vary much from that shown in normal subjects. The highest T waves were seen in the febrile cases. The average height of the T waves in the febrile cases was 4.8 millivolts and in the afebrile 3.6 millivolts. The height of this wave ranged from 2 to 8 millivolts (table 9).

TABLE 9.—*T Wave*

		Scarlet Fever		
	Normal	Average	Febrile	Afebrile
Highest	8.0 mv.	8.0 mv.	8.0 mv.	7.0 mv.
Lowest	1.5 mv.	2.5 mv.	2.5 mv.	2.0 mv.
Average	3.5 mv.	4.0 mv.	4.8 mv.	3.6 mv.

Abnormalities in the shape and direction of the T wave in leads 1 and 2 were noted in only 10 per cent of the children. Inversion of the T wave was noted in all leads in only one case (fig. 5). Changes in the R-T interval were also seen in this child. These conditions all cleared up before the patient was discharged from the hospital. Rounded, widened, iso-electric or low voltage T waves were observed in four cases (fig. 6). These abnormalities were seen in the early days of the disease and all returned to normal before the patient was discharged. Disturbances in the shape and the height of the T waves were considered abnormal only when these conditions changed during the course of the disease.

In 40 per cent of these children an inversion of the T wave in the third lead was noted at some time during the course of the disease (figs.

23. Rothschild, M. A.; Sacks, B., and Libman, B.: Disturbances of the Cardiac Mechanism in Subacute Bacterial Endocarditis and Rheumatic Fever, Am. Heart J. **2**:363, 1926-1927.

3, 4 and 8). In 36 per cent of the children T^3 changes only were found. The direction of the T wave changed during the course of the disease. In view of our observations in normal children, we did not consider this as evidence of myocardial involvement, although it occurred more frequently in scarlet fever (figs. 3 and 4).

T-P INTERVAL

The average T-P interval for normal children was 0.19 second and for children with scarlet fever, 0.185 second. The average in the febrile stage of scarlet fever was 0.16 second and in the afebrile, 0.22 second.

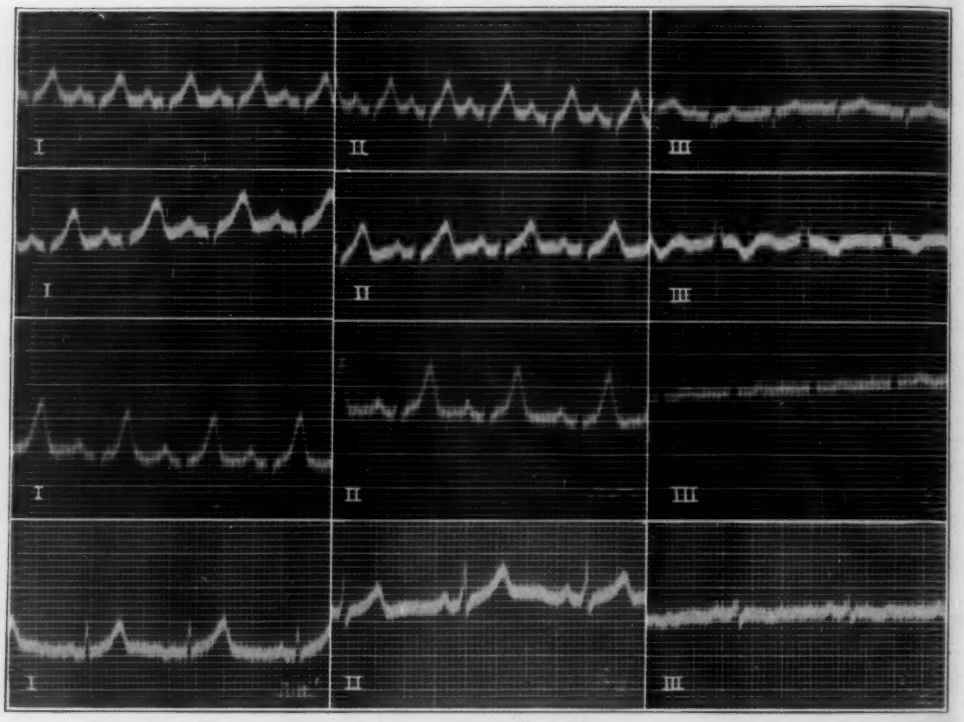

Fig. 8.—Tracings in the illness of a child, aged 6 years. These tracings were taken on the sixth day of the illness (upper row, temperature 101.0 F.), and on the ninth (second row, 100.4 F.), fourteenth (third row, 100.0 F.) and twenty-third (fourth row, 99.6 F.) days. This plate illustrates the occurrence of a left axial deviation which returned to normal on the twenty-third day of disease. Note here also the changes in the T wave on the various days, most marked inversion on the ninth day of illness when a definite left axial deviation is seen. The left axial deviation is already noticeable on the sixth day of illness. It is most marked on the ninth day and on the twenty-third day, one sees a normal axial deviation.

RELATIONSHIP OF P-T TO P-P

In children with scarlet fever the relationship of the length of the systolic portion of the electrocardiographic curve to the length of the

whole cardiac cycle was exactly the same as that in normal children. The average length of the P-T interval was 0.45 second. The average length of the whole cycle was 0.634 second. The ratio was 70.8 per cent.

DEVIATION OF THE AXIS

In 16 per cent of the cases, an abnormal axial deviation was noted during the second week of the illness: in 12 per cent, a left axial deviation (figs. 7 and 8) and in 4 per cent a right axial deviation (fig. 4). In all but one case this abnormality returned to normal before the patient was discharged. McCulloch [24] observed frequent changes in deviation of the axis, particularly to the left, in clinical diphtheria, and believed that it was a sign of failure of either the right or left ventricle. Nathanson [25] observed a definite change in axial deviation within forty-eight

TABLE 10.—*Deviation of the Axis*

Left Axial Deviation 4 per Cent. Right Axial Deviation 12 per Cent. Persists 2 per Cent

Patient	Age, Years	Axial Deviation	Day of Appearance	Return to Normal	Other Changes	Bradycardia
D.D.....	7	Left	4th	6th day	T^3 inverted	No
F.W.....	6	Right	2nd	7th day	T^2 and T^3 changes	Yes
N.W.....	7	Right	9th	21st day	T^3 changes	No
O.I.....	12	Right	6th	13th day	T^2 changes	Yes
P.S.....	8	Right	4th	7th day		No
V.L.....	11	Right	13th	17th day		No
S.H.....	6	Right	4th	Persists		Yes
W.H.....	11	Left	4th	7th day	R-T changes	Yes

hours following the injection of diphtheria toxin in animals. There seems to be no relationship in our cases between the occurrence of bradycardia, abnormalities in the ventricular portion of the electrocardiogram and the occurrence of axial deviation. Of the eight cases showing this disturbance, only two showed disturbances in the ventricular portion of the electrocardiogram and in four a relative bradycardia developed (table 10). We are not prepared to state what is the exact significance of these disturbances. The disturbances may be due to an intracardiac parenchymal disease, or they may be due to infradiaphragmatic disturbances. We have no evidence for either supposition.

COMMENT

The electrocardiographic abnormalities found during the course and convalescence in scarlet fever differ from those described in rheumatic fever. 1. No disturbances in conduction were observed. 2. Only

24. McCulloch, H.: Studies of Effect of Diphtheria on the Heart, Am. J. Dis. Child. **20**:89 (Aug.) 1920.

25. Nathanson, M. H.: Electrocardiogram in Diphtheria, Arch. Int. Med. **42**:41 (July) 1928.

minimal disturbances of the ventricular portion of the electrocardiogram were noted, minimal both in frequency of occurrence and in the severity of the abnormality. 3. Changes occurred as a rule early in the disease and disappeared before the discharge of the patient from the hospital. The bradycardia and the axial deviation have already been discussed.

It seems to us that whatever changes take place in scarlet fever occur early in the course of the disease and, while they are evidence of a myocardial disturbance, the disturbance must apparently be a toxic parenchymal one of mild degree. These abnormalities not only do not persist, but they disappear as a rule in the early days of the illness.

SUMMARY

1. Fifty patients with scarlet fever admitted consecutively were studied.

2. The cardiac rate did not differ from that in normal children in the same age group. In children with fever the increase in rate was commensurate with the increase in temperature.

3. A relative bradycardia, beginning in the second week and returning to normal at the end of the third week, was observed in 25 per cent of the cases.

4. Sinus arrhythmia occurred just as frequently as it did in normal children.

5. No prolongations of the P-R interval were observed.

6. Only minor changes in the T or the R-T waves were noted in 10 per cent of the children. These changes returned to normal before the patient was discharged.

7. An abnormal axial deviation occurring during the course of the illness was noticed in 16 per cent of the children. The deviation of the axis returned to normal limits before discharge in all but one child.

8. These observations differ from those described in rheumatic fever in (*a*) the comparative infrequency of electrocardiographic evidence of myocardial involvement; (*b*) the absence of any tendency for abnormalities, when present, to persist, and (*c*) the absence of prolongation of the P-R interval.

HUMAN MILK STUDIES

VII. CHEMICAL ANALYSIS OF MILK REPRESENTATIVE OF THE ENTIRE FIRST AND LAST HALVES OF THE NURSING PERIOD *

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AND

HELEN A. HUNSCHER, B.S.

DETROIT

The milk secreted by women has been shown to vary in composition between wide limits,[1] and it is difficult to define a standard composition of milk that will adequately supply the nutritive needs of all growing infants. Observations have shown that breast milk may vary considerably from time to time as the constituents change in concentration throughout the progression of the period of lactation. It may vary from day to day, at different times of the day and night and during a single nursing period. However, individuality in the composition of the milk of the individual woman tends to persist. Furthermore, a study of the internal and external factors influencing the secretion of human milk indicates that certain functions may have a bearing on the changes in the composition of the milk itself. It has been demonstrated that the following conditions may be effective in altering the quality and quantity of the milk secreted: the size and anatomic structure of the

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* From the Nutrition Research Laboratories of the Merrill-Palmer School and the Children's Hospital of Michigan, now the Research Laboratories of the Children's Fund of Michigan.

* The term "nursing period" is used for a single feeding of the baby and "lactation period" for the whole duration of lactation.

† Miss Minerva Brown died in 1929. During the later progress of these studies, she had pursued graduate study elsewhere.

1. Talbot, F. B.: A Summary of Present Knowledge of Human Milk, Am. J. Dis. Child. **7**:445 (June) 1914. Gardner, J. A., and Fox, F. W.: On the Variations in the Composition of Normal Human Milk, Practitioner **114**:153, 1925. Myers, B.: An Investigation on the Analysis of Human Milk, Brit. J. Child. Dis. **24**:249, 1927. Bell, M.: Studies on the Composition of Human Milk, J. Biol. Chem. **80**:239, 1928.

mammary glands;[2] the amount and kind of food ingested;[3] the environmental conditions, such as fresh air, sunshine and the amount and intensity of work, rest and exercise;[4] the emotional and psychic make-up of the person, and also extraneous disturbances.[5]

It is evident from this review that there are many influences operating to change not only the total quantity of breast milk secreted, but also its integral parts. Over and above these facts, how significant are the variations in the composition of milk in the well-being of the average breast-fed infant? Does the maternal physiologic mechanism of the individual mother provide a changing composition of milk to take care of her child, or, if not, are these fluctuations chaotic and grossly beyond the margin of safety of the infant, thus being to his detriment? Detailed chemical studies of human milk from the same women collected throughout the nursing period, from each breast, at regular intervals of the day and night and from day to day, month to month and lactation to lactation, might yield information contributory to the solution of the great enigma of the physiology of the secretion of human milk, and consequently might add to our knowledge of the nutrition of infants.

In view of the fact that a vast amount of experimental work has been done on the physiology of the secretion of milk in the animal industry, it seems advantageous and pertinent to review some of the results obtained in that field as a background for the data that are to be recorded in this report and others that are to follow on the chemistry of human milk.

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2. Macy, I. G., and Outhouse, J.: Breast Milk: A Variable Food, J. Am. Dietet. A. **4**:9, 1928. Macy, I. G.; Hunscher, H. A.; Donelson, E., and Nims, B.: Human Milk Flow, Am. J. Dis. Child. **39**:1186 (June) 1930.

3. Hoobler, B. R.: The Effect on Human Milk Production of Diets Containing Various Forms and Quantities of Protein, Am. J. Dis. Child. **14**:105 (Aug.) 1917. Grumme, F.: Ernahrung und Laktation, Monatschr. f. Geburtsh. u. Gynäk **53**:271, 1920. Courtney, A. M.: The Effect of Inadequate Diet on the Inorganic Salt Content of Mother's Milk, Am. J. Dis. Child. **26**:534 (Dec.) 1923. Adair, F. L.: The Influence of Diet on Lactation, Am. J. Obst. & Gynec. **9**:1, 1925. Kleiner, I. S.; Tritsch, J. E., and Graves, L. G.: The Influence of Supplementary Feeding of Carbohydrate upon Lactation, Am. J. Obst. & Gynec. **15**:172, 1928. Macy, I. G. et al.: A series of papers on the quality and quantity of food consumed by lactating women, in press, J. Nutrition.

4. Macy and Outhouse (footnote 2, first reference). Rotch, T. M.: Pediatrics; The Hygienic and Medical Treatment of Children, ed. 3, Philadelphia, J. B. Lippincott Company, 1901. Morse, J. L.: Clinical Pediatrics, Philadelphia, W. B. Saunders Company, 1926.

5. Macy and Outhouse (footnote 2, first reference). Hunscher, H. A.; Vincent, E. L., and Macy, I. G.: Psychophysiological Studies. 1. The Technique of Securing Quantitative and Coordinated Psychological and Physiological Data on Lactating Women in Their Usual Home Environment, J. Child Development **1**: 15, 1930.

Fluctuations in the composition of milk have been observed in the cow and the goat, as well as in the human being, and numerous experiments on the actual rate of the formation of milk, glandular activity and composition of the mammary gland have given rise to several theories concerning these fluctuations.

Taylor and Husband [6] suggested that the percentages in the composition of cow's milk seem to be determined by its rate of flow. Much controversy has arisen concerning the actual rate of secretion of milk in the interim and during a milking period. It has been generally thought that the most rapid elaboration occurs at the actual time of milking, since the gland would be incapable of storing the total quantity obtained at one milking.[7] Other experiments have indicated that not only the total quantity of milk, but also an amount in excess of that secured on milking, may be present at one time in the udder.[8]

If the composition of milk is dependent on its rate of secretion, which is either accelerated or retarded during the process of expression, changes in the composition may be expected. Concerning the rate of secretion and the relative composition resulting, Cockefair [9] said:

> What is meant by secretion? Either it is liberated from the alveolar cells, or synthesized, or it is stored in the cell vacuoles until the stimulus of milking sets in motion the reflexes which produce rapid release of the products.

This work supported the preceding evidence of Ragsdale, Turner and Brody [10] that the rate of elaboration of the constituents in milk is inversely proportional to the quantity of constituents accumulated. They concluded that the formation of milk is most active immediately after milking and proceeds in diminishing rate as the pressure and presence of stored milk interfere with cellular activity. This theory is further

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6. Taylor, W., and Husband, A. D.: The Effect on the Percentage Composition of the Milk of (*a*) Variations in the Daily Volume; (*b*) Variations in the Nature of the Diet, J. Agric. Sc. **12**:111, 1922.

7. Isaachsen, H.: Some Aspects of the Physiology of the Mammary Gland, Proc. World Dairy Congress **2**:1021, 1923; quoted by Petersen, Palmer and Eckles: Am. J. Physiol. **90**:573, 1929.

8. Zwart, S. G.: Ztschr. f. Fleisch- u. Milchhyg. **26**:231, 1916; quoted by Petersen, Palmer and Eckles (footnote 7). Gaines, W. L., and Sanmann, F. P.: The Quantity of Milk Present in the Udder of the Cow at Milking Time, Am. J. Physiol. **80**:691, 1927. Gowen, J. W., and Tobey, E. R.: Significance of the Chemical Composition of the Secreting and Dry Mammary Gland to Milk Secretion, J. Gen. Physiol. **12**:123, 1928.

9. Cockefair, E. A.: The Activity of the Mammary Gland as Determined by Analysis of the Mammary Gland before and after Milking, J. Dairy Sc. **11**:230, 1928.

10. Ragsdale, A. C.; Turner, C. W., and Brody, S.: The Rate of Milk Secretion as Affected by an Accumulation of Milk in the Mammary Gland, J. Dairy Sc. **7**:249, 1924.

supported by evidence from studies on lactating women conducted in these laboratories. More frequent removal of the accumulated lacteal products from three to six times daily resulted in increased cellular activity as measured by the augmented output of milk.[11]

Such studies on glandular capacity and the rate of the flow of milk seem pertinent in discussing the results of other experiments. Our meager knowledge of the processes involved in the formation of milk makes it necessary to know the relative time at which the activities of actual formation and release of products take place. Consequently, with a view to learning the manner of its formation, studies have been made of the individual components of milk,[12] as well as of the quantity of milk secreted. Since milk fat was considered by Petersen, Palmer and Eckles [13] to be an important ingredient, varying considerably, they made a series of observations on its mode of secretion and on the basis of their observations suggested that the formation of fat is governed by an entirely separate mechanism than that governing the formation of other constituents. Chemical analyses of the udders of cows have been made from time to time with the object of learning more about the method involved in the synthesis of the individual components of milk.[14] Gains,[15] who made a statistical study of the results of chemical determinations of cow's milk, suggested:

> If any two constituents are intimately related in the mechanism of milk secretion, so that the rate of secretion of one increases the rate of the other accordingly, then values of these two expressed in percentage composition of the various samples of milk, will tend to bear to each other a constant ratio.

From these citations it is apparent that more complete knowledge concerning the processes of the secretion of milk in both the cow and

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11. Macy et al. (footnote 2, second reference).

12. Porcher, C.: Ueber die Entstehung des Milchzuckers, Biochem. Ztschr. **23**:370, 1910. Paton, D. N., and Cathcart, E. P.: On the Mode of Production of Lactose in the Mammary Gland, J. Physiol. **42**:179, 1911. Cary, C. A.: Amino-Acids of the Blood as the Precursors of Milk Proteins, J. Biol. Chem. **43**:477, 1920. Editorial: The Synthetic Powers of the Mammary Gland, J. A. M. A. **72**:864 (March 22) 1919. Meigs, E. B.: Milk Secretion as Related to Diet, Physiol. Rev. **2**:204, 1922. Wright, N. C.: The Mechanism of Secretion of Calcium and Phosphorus in Milk, J. Agric. Sc. **18**:478, 1928.

13. Petersen, W. E.; Palmer, L. S., and Eckles, C. H.: The Synthesis and Secretion of Milk Fat: I. The Time of Milk and Fat Secretion, Am. J. Physiol. **90**:573, 1929.

14. Gowen and Tobey (footnote 8, third reference). Laxa, O.: Chemical Composition of the Mammary Gland of the Cow, Chem. Abstr. **23**:891, 1929; Lait **7**:336, 1927. Petersen, W. E.; Palmer, L. S., and Eckles, C. H.: The Synthesis and Secretion of Milk Fat: II. An Analytical Study of the Fat of the Bovine Mammary Gland, Am. J. Physiol. **90**:582, 1929.

15. Gaines, W. L.: Relative Rates of Secretion of Various Milk Constituents, J. Dairy Sc. **8**:486, 1925.

the human mother is needed. Moreover, it would seem of nutritional and clinical importance to know the significance of the differences in composition of the milk that the infant obtains at the first or the last part of nursing, from either breast and at different times of the day and night. The present report on the chemical composition of milk expressed during the first and the last halves of the nursing period at continuous intervals of four hours during a twenty-four hour day is the first of a series of papers from these laboratories on the chemistry of human milk.

EXPERIMENTAL PROCEDURE

Subjects.—The women of this investigation lived with their own families in their usual environments during the periods of observation. Histories of the cases are recorded in table 1.

TABLE 1.—*Histories of Cases in Women under Observation*

	Subject 6	Subject 7	Subject 9
Descent	Dutch, French, German, Danish	Scotch, Irish, American	German
Age	25 years	33 years	23 years
Height	62.2 inches	68.5 inches	66 inches
Body weight	64.9 Kg.	(a) 65.8 Kg. (b) 64.9 Kg.	60.6 Kg.
Usual physical state	Apparently healthy; persistently low hemoglobin	Healthy; no defects	Healthy
Number of pregnancies	Three	Two	One
Date of birth of babies	Oct. 13, 1926	Nov. 20, 1926	Sept. 2, 1930
Date of study	March 15, 1927	(a) March 3, 1927 (b) April 25, 1927	(a) Nov. 5, 1930 (b) Nov. 25, 1930
Weight of infant at birth	7 lb., 8 oz.	8 lb., 3 oz.	8 lb., 10 oz.
Weight of infant at time of study	17 lb., 12 oz.	(a) 16 lb., 6 oz. (b) 18 lb.	(a) 11 lb., 15 oz. (b) 12 lb., 14 oz.
Development of infant	Excellent	Excellent	Excellent (slight eczema)
Average output of milk per 24 hour period	2,600 cc.	2,000 cc.	584 cc.

Method of Collecting the Milk.—The many data recorded in the literature by various investigators have been obtained under widely differing conditions and methods of collecting, sampling and analyzing milk. The great dissimilarity in the defining and sampling of beginning and end milk may explain some of the widely divergent results. In this study, an attempt has been made to obtain for analysis standardized samples that could be considered representative of the milk during the first and last halves of a nursing period.

Preliminary observations were made on the total volume manually expressed from each breast at continuous nursing intervals of four hours during a twenty-four hour day immediately preceding the day of the study. From such measurements it was possible to predict the approximate volume of the output of milk from each breast at the various nursing periods. A third of the volume of milk expressed from a single breast at a specific period was collected in the first graduated cylinder, the intermediate quantity in a second, and the remainder or strippings in a third. In this way the milk was carefully measured and the intermediate portion divided so that the contents of the first and third containers were of equal volume, thus representing the first and the last halves of the milk

from this breast at a specific nursing period. This procedure was used in obtaining samples from both breasts at the nursing interval. The specimens of the first half from the two breasts were pooled and analyzed by approved chemical methods and likewise those from the last half, giving a representative sampling of the entire first and last halves of a single nursing period. This standardized procedure was followed for every interval of four hours throughout the day and night.

TABLE 2.—*Illustration of Method of Sampling Milk from the First and Last Halves of a Nursing Period from the Two Breasts at Intervals of Four Hours* in the Twenty-Four Hour Day*

Observation		Volume of Milk Manually Expressed										Combined Milk, Cc.
		Left Breast, Cc.					Right Breast, Cc.					
Subject	Time	Begin	Interval	End	Total	Each Half	Begin	Interval	End	Total	Each Half	Each Half
7 Normal baby	10 a.m.	75	76	69	220	110	39	43	57	139	70	180
	2 p.m.	95	93	55	343	172	86	84	94	264	132	304
	6 p.m.	79	79	51	209	105	74	72	47	193	97	202
	10 p.m.	81	79	58	218	109	68	68	64	200	100	209
	2 a.m.	83	83	85	251	126	48	48	90	186	93	219
	6 a.m.	100	102	50	252	126	82	82	23	187	94	220
	Total 24 hr.				1,493					1,169		
6 Normal baby	10 a.m.	63	60	97	220	110	73	73	116	262	131	241
	2 p.m.	75	73	61	209	105	87	88	93	268	134	239
	6 p.m.	97	97	79	273	137	109	109	83	301	151	288
	10 p.m.	90	91	79	260	130	110	112	89	311	156	286
	2 a.m.	81	81	58	220	110	99	99	67	265	133	243
	6 a.m.	87	89	93	269	135	103	104	67	274	137	272
	Total 24 hr.				1,451					1,681		
9 (a) Baby with eczema	10 a.m.	27	27	0	54	27	10	11	35	56	28	55
	2 p.m.	20	19	6	44	22	20	20	0	40	20	42
	6 p.m.	17	17	15	68	34	15	15	5	34	17	51
	10 p.m.	12	10	54	76	38	18	18	23	59	30	68
	2 a.m.	17	17	14	68	34	21	21	11	53	27	61
	6 a.m.	13	14	30	56	28	20	20	28	68	34	62
	Total 24 hr.				366					310		
9 (b)	10 a.m.	26	27	14	67	33	12	10	24	46	23	56
	2 p.m.	18	21	3	42	21	20	21	14	55	27	48
	6 p.m.	17	15	23	55	27	15	14	8	37	18	45
	10 p.m.	11	11	13	35	14	17	17	12	46	23	37
	2 a.m.	17	16	4	37	18	21	3	0	24	12	30
	6 p.m.	13	15	24	52	26	19	17	0	36	18	44
	Total 24 hr.				288					244		

* The breasts were stripped at 6 a.m.; therefore the sample represents that which was secreted between 6 and 10 a.m.

In Table 2 are shown the times of nursing, the volume of milk from each breast, at each of the three intervals, and the combination of these to form representative samples of equal volume of the first and last halves of the nursing period.

Methods of Analysis Used.—The following approved chemical methods were used in this particular study: the micro-Babcock[16] method for fat; the A. O. A. C.[17] method for total solids; 50 or 75 cc. of milk ashed in a platinum crucible

16. Hawk, P. B., and Bergeim, O.: Practical Physiological Chemistry, ed. 9, Philadelphia, P. Blakiston's Son & Co., 1927.

17. Official and Tentative Methods of Analysis of the Association of Official Agricultural Chemists, ed. 2, Washington, D. C., 1925, vol. 16.

at approximately 450 C. for total ash; the method of Kjeldahl[16] for total nitrogen; the A. O. A. C.[17] method 1 for separation and determination of casein and albumin; 25 cc. of a tungstic acid filtrate[18] for the determination of the total nonprotein nitrogen according to the method of Kjeldahl; the method of Folin[18] for amino-acids, using 2 cc. of sodium carbonate instead of a few drops to counteract the buffer action of lacteal salts, and a standard of 0.07 mg. of glycin-nitrogen; the method of Shaffer and Hartmann[19] for lactose; Clark's[20] method for calcium; the method of Fiske and Subbarow[21] for phosphorus, and Van Slyke's[22] method for chlorides.

RESULTS

The magnitude of change in the composition of milk occurring between the beginning and the end of a nursing period, as recorded from the literature, has been summarized in table 3. The early reports are of interest, even though they carry figures on breast milk obtained from few cases and under heterogeneous methods of collection and chemical analysis and the range of percentage of differences between corresponding determinations is extreme, as might be expected. These results, however, cannot be compared directly with those of later investigators who used more reliable methods. Furthermore, none of them is analogous to the data to be presented herein, which represent the complete first and last halves of the milk secreted over definite intervals of time, during the day and night, and therefore are not comparable to samples of milk expressed before the baby is allowed to nurse and thereafter (strippings).

The fact that variations in the compostion of human milk occur during the progress of a nursing period has been noted frequently in the literature, but the knowledge regarding the extent or nature of these changes is meager and inconclusive, owing more or less to unstandardized conditions and methods of sampling and analyzing milk.

It is uniformly shown that the lacteal fat increases from the beginning to the end of nursing. The summary of published analyses (table 3) shows that the magnitude of the percentage of increase varies considerably, the values ranging between 6 and 79 per cent. In analyses

18. Folin, O. K.: Laboratory Manual of Biological Chemistry, with supplement, ed. 3, New York, D. Appleton and Company, 1922.

19. Shaffer, P. A., and Hartmann, A. F.: Methods for the Determination of Reducing Sugars in Blood, Urine, Milk, and Other Solutions: II. The Iodometric Determination of Copper and Its Uses in Sugar Analysis, J. Biol. Chem. **45**:365, 1921.

20. Clark, G. W.: The Micro Determination of Calcium in Whole Blood, Plasma, and Serum by Direct Precipitation, J. Biol. Chem. **49**:487, 1921.

21. Fiske, C. H., and Subbarow, Y.: The Colorimetric Determination of Phosphorus, J. Biol. Chem. **66**:375, 1925.

22. Van Slyke, D. D.: The Determination of Chlorides in Blood and Tissues, J. Biol. Chem. **58**:523, 1923.

TABLE 3.—*Percentage of Various Constituents in Breast Milk Collected at the Beginning and End of Nursing as Compiled from Available Literature*

Author	Date	No. of Determinations	Fat			Lactose			Protein			Total Solids			Total Ash		
			Begin	End	Percentage of Difference*	Begin	End	Percentage of Difference	Begin	End	Percentage of Difference	Begin	End	Percentage of Difference	Begin	End	Percentage of Difference
Reiset, cited by Thiemich: Monatschr. f. Geburtsh. u. Gynäk. **8**: 521, 1898; **9**: 504, 1899	1849	3	....	....	....	....	....	....	....	....	....	10.58 12.78 13.46	12.93 15.52 14.57	+18 +18 + 8			
Mendes de Leon, cited by Thiemich	1881	Average 9	2.62	6.03	+57	....	....	....	....	....	....	9.38	13.23	+29			
Pfeiffer, cited by Thiemich.....	1883	2	0.81 3.09	3.81 5.08	+79 +41	5.98	6.38	+ 6	1.43 1.80	1.33 1.71	— 7 — 5	11.55	13.33	+13	0.161	0.137	—15
Johannessen, cited by Judson and Gittings: The Artificial Feeding of Infants, Lippincott, 1902	1892	1	2.77	3.94	+30	5.70	5.09	—11	0.98	0.95	— 3						
Forster: cited by Judson and Gittings	....	1	1.70	4.51	+62	5.56	5.10	— 8	1.13	0.71	—37	.....	.....	....	0.46	0.28	—39
Baumm and Illner, cited by Thiemich	1894	1	4.11	4.39	+ 6	6.32	6.80	+ 7	2.01	2.04	+ 2	12.60	13.39	+ 6	0.160	0.160	0
Michel, cited by Thiemich.....	1897	3	2.48 4.58 0.96	3.70 4.84 2.14	+33 + 5 +55	....	....	....	1.24 1.81	1.57 1.77	+21 — 2	.....	.....	....	0.150 0.240	0.166 0.200	+10 +17
Guiraud, cited by Thiemich.....	1897	3	1.75 1.80 1.90	5.18 6.18 5.98	+66 +71 +68	8.07 7.92 8.18	7.19 8.07 7.79	—11 + 2 — 4	0.9 1.2 0.95	1.1 1.4 1.2	+18 +14 +21	....	....	....	0.16 0.20 0.18	0.19 0.22 0.18	+16 + 9 0
Adriance and Adriance, cited by Thiemich	1897	3	3.66 1.98 1.19	5.11 3.12 2.78	+26 +36 +55	6.82 6.56 7.00	6.92 7.11 6.93	+ 1 + 8 — 1	1.45 0.58 0.56	1.00 0.72 0.95	—31 +20 +41	12.11 9.26 8.95	13.25 11.14 10.79	+ 8 +16 +17	0.16 0.12 0.15	0.20 0.17 0.12	+20 +29 —20
Carter and Richmond, cited by Judson and Gittings	1898	Average 37	2.89	3.18	+ 9	6.51	6.53	+ 0.3	1.99	1.99	0	.....	.....	....	0.28	0.26	— 7
Denis and Talbot: Am. J. Dis. Child. **18**: 93, 1919	1919	Average 56	3.79	6.19	+39	7.10	6.02	—15	1.43	1.47	+ 3						
Wittenberg: Monatschr. f. Kinderh. **32**: 510, 1926	1926	Average 7	....	....	....	5.36	6.29	+15									
Myers: Brit. J. Child. Dis. **24**: 249, 1927	1927	Average	3.48	4.98	+30	5.67	5.64	— 1	1.36	1.49	+ 8	.....	.....	....	0.20	0.20	0
Range of percentage of difference*.....			+6 to +79			—15 to +15			—31 to +41			+6 to +29			—39 to +29		

* Percentage of increase at end of nursing = + per cent; percentage of decrease at end of nursing = — per cent.

of milk collected under standardized methods, as shown in table 4, an augmentation of fat occurred in the last half of the nursing period, as demonstrated by the trend of the percentage of differences. The medium of the percentage of differences, an increase of 26 per cent, illustrates the central tendency. The magnitude of difference is not as great in the present study as in some of the published reports, owing to the fact that the specimens used in the study represent the whole of the milk expressed in the first and the last halves, and not a small fraction at the beginning and the strippings.

Are variations in the composition of the milk secreted in the first and last intervals of the nursing period regulated by actual changes in the mechanism of secretion, by mechanical influences of pressure in suckling or expressing, or are they indefinite variations? Based on the theories of the secretion of milk, the increased fat, total solids, ash and nitrogen observed in the milk in the later part of the milking period have been attributed to a progressively rapid release of the cellular products as the activity of the cells increased with the mechanical stimulus of milking. Various authors have expressed the belief that the increase of fat in the later interval of milking is due to a physical retardation and obstruction in the passage of fat through the ducts and fine spaces of the gland, which by increased mechanical pressure of milking would be squeezed out in the last portion.[23] On the contrary, the experiments of Petersen, Palmer and Eckles,[24] in which a microscopic examination of the excised udder revealed a normal distribution of large and small globules of fat, gave evidence against the theory of physical retardation of fat in the ducts.

Concerning changes in human milk, Levy[25] suggested that variations are regulated by the condition of the nursing infant. The milk "changes not only several times a day and during each nursing, but is influenced and determined by the frequency of nursing, length of nursing, and vigor of nursing, that is, it adjusts itself to the kind of baby and the condition of the baby at the breast."

A study undertaken by Smith and Merritt[26] to determine the rate at which the baby secures milk from the breast brought out the fact

23. Gaines (footnote 15); Swett, W. W.: Relation of Conformation and Anatomy of the Dairy Cow to her Milk and Butter Fat Producing Capacity, Udder Capacity and Milk Secretion, J. Dairy Sc. **10**:1, 1927. Marshall, F. H. A.: The Physiology of Reproduction, ed. 2, New York, Longmans, Green & Co., 1922, p. 590.

24. Petersen, W. E.; Palmer, L. S., and Eckles, C. H.: The Synthesis and Secretion of Milk Fat. III. A Study of the Activity of the Perfused Surviving Gland with Special Reference to the Fat, Am. J. Physiol. **90**:592, 1929.

25. Levy, J.: Adaptability of Breast Milk, J. A. M. A. **72**:1285 (May 3) 1919.

26. Smith, C. H., and Merritt, K. K.: The Rate of Secretion of Breast Milk, Am. J. Dis. Child. **24**:413 (Nov.) 1922.

TABLE 4.—*Solid Constituents* *

Observations		Fat			Lactose			Protein			Total Solids			Solids Not Fat		
Subject	Time	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference
7 (a)	10 a.m.	4.12	5.59	+26	6.98	7.25	+ 3.7	1.03	1.02	—1.0	12.37	13.14	+ 6	8.25	7.55	— 9.1
	2 p.m.	3.85	5.00	+23	7.24	7.13	— 1.5	1.02	1.03	+1.0	11.15	12.34	+10	7.30	7.34	+ 0.5
	6 p.m.	3.91	6.07	+36	6.87	6.90	+ 0.4	1.04	1.08	+3.7	11.46	12.50	+ 8	7.55	6.43	—14.8
	10 p.m.	3.50	6.20	+44	7.14	7.20	+ 0.8	0.94	0.98	+4.1	11.31	12.38	+ 9	7.81	6.18	—20.9
	2 a.m.	2.83	3.81	+26	7.39	7.19	— 2.7	0.96	0.99	+3.1	10.81	11.62	+ 7	7.98	7.82	— 2.0
	6 a.m.	1.34	1.85	+28	7.22	7.22	0.0	0.92	0.99	+7.1	11.11	12.05	+ 8	9.77	10.20	+ 4.2
7 (b)	10 a.m.	3.40	4.38	+22	7.13	7.34	+ 2.9	1.09	1.08	—0.9	11.72	12.34	+ 5	8.32	7.96	— 4.3
	2 p.m.	3.26	4.18	+22	7.15	7.19	+ 0.6	1.11	1.10	—0.9	11.37	12.30	+ 8	8.11	8.12	+ 0.1
Range of percentage of difference				+22 to +44			—2.7 to +3.7			—1.0 to +7.1			+5 to +10			—20.9 to +4.2
Average percentage of difference				+28			+0.6			+2.1			+8			—5.8
6	10 a.m.	5.87	8.29	+29	7.18	7.15	— 0.4	1.04	1.07	+2.8	12.94	14.84	+13	7.07	6.55	— 7.4
	2 p.m.	4.96	6.32	+23	7.24	7.37	+ 1.8	1.11	1.13	+1.8	11.89	12.90	+ 8	6.93	6.58	— 5.1
	6 p.m.	3.35	5.88	+43	7.11	7.10	— 0.1	1.11	1.16	+4.3	11.91	13.54	+12	8.56	7.66	—10.5
	10 p.m.	3.81	5.18	+26	7.32	6.98	— 4.6	1.04	1.05	+0.9	11.85	12.96	+ 9	8.04	7.78	— 3.2
	2 a.m.	3.66	4.63	+21	7.20	7.12	— 1.1	1.08	1.11	+2.7	11.60	12.51	+ 7	7.94	7.88	— 0.8
	6 a.m.	3.79	4.68	+19	7.24	7.21	— 0.4	1.04	1.10	+5.5	11.70	12.77	+ 8	7.91	8.09	+ 2.2
Range of percentage of difference				+19 to +43			—4.6 to +1.8			+0.9 to +5.5			—7 to —13			—10.5 to +2.2
Average percentage of difference				+27			—0.8			+3.0			—10			—4.1
9 (a)	24 hr.	5.6	6.26	+11	6.39	7.82	+18.3	1.12	1.09	—2.7	12.74	13.92	+ 8	7.14	6.66	— 7.0
9 (b)	24 hr.	4.56	6.56	+31	7.21	7.81	+ 7.7	1.05	1.06	+0.6	12.71	14.20	+11	8.15	7.64	— 6.3
Median of percentage of differences				+26			+0.2			+2.4			+8			—4.7

* The amounts are given in grams per hundred cubic centimeters of milk.

that the total quantity (and therefore the total amount of various constituents) obtained in a certain length of time was dependent on the weight and size of the baby. Most of the milk (from 65 to 80 per cent) was obtained in the first four minutes, the average time for emptying being from six and one-half to seven and one-half minutes. However, a few large, vigorous infants obtained small amounts of milk up to sixteen minutes, and probably a milk usually designated as strippings.

From these studies the practical significance of learning the extent of changes in the milk obtained by the infant during the first two minutes and during the remaining time is obvious. If the milk tends to become more concentrated, as has been observed clinically and reported in the literature, the strong, large infant would perhaps obtain not only a greater amount but also a more concentrated grade of milk, and, on the other hand, the underweight, weak baby might be less fortunate and obtain a milk inferior in both quantity and quality.

Czerny and Keller [27] concluded from their studies that wide variations in the fat of milk cause nutritional disturbances in infants, and Koeppe [28] further emphasized the clinical value of determining the relative percentages of fat in the milk the infant receives in relation to his weight and general development.

Clinical observations have furnished the incentive for more detailed chemical studies of the milk expressed from the breast, representative of the first and the last half of the nursing sample that the baby would receive. It has been brought to our attention by Dr. Emmett C. Troxell, dermatologist, that in some vigorous, hardy, breast-fed infants a certain type of eczema develops that may be cleared up by shortening the nursing period and allowing the infant to nurse only two or three minutes from each breast, thus permitting him to take the first portion of the milk and not the strippings. This would approximate the first half of the output from each breast, or 50 per cent, according to the observations of Smith and Merritt.[26] A further illustration of the need for more extensive knowledge of the chemistry of breast milk as it relates to the well-being of the infant is the demonstration of improved growth and development of some weak and undernourished babies when they are allowed to secure the last portion of the milk in the mammary gland, the first portion having been removed by manual expression or by breast pump.[28]

27. Czerny, A., and Keller, A.: Des Kindes Ernährung, Ernährungsstörungen und Ernährungstherapie, ed. 1, Leipzig, Franz Deuticke, 1909.

28. Koeppe, H.: Ueber den klinischen Wert der Fettbestimmung der Frauenmilch, Jahrb. f. Kinderh. **5**:177, 1924.

Lacteal fat, beyond supplying the immediate needs of the baby, has been said to favor mechanically the digestion of protein,[29] a fact that emphasizes the importance of the proper proportion of fat and protein in the milk. The significance of the proper amount of fat in the maintenance of normal conditions of elimination has been emphasized by Griffith [30] and Rotch.[31] Since the digestibility of lacteal fat seems to be closely associated with the degree of coagulation of casein,[32] the analysis of protein in the milk at the beginning and the end of the nursing period is of further interest.

Published figures on the total protein content of breast milk at the beginning and at the close of nursing are summarized in table 3. Notable fluctuations occur to the extent of a difference of from —37 to + 21 per cent. No doubt such irregularities are due to the methods of sampling and analyzing the milk. On the other hand, the percentage of differences in the protein content of milk representative of the first and the last halves of the milk expressed, as shown in table 4, demonstrates likewise that the protein content of the milk may tend to increase as the nursing period progresses, but in less degree than is the case with the fat.

Protein has often been designated as one of the factors causing digestive disturbances in infants. This fact has led to comparative studies of the various nitrogen constituents in the milk in relation to the nutrition of breast-fed infants. The proportional amounts of albumin and casein protein in human milk have been reported to be definite factors in the nutrition of the infant. This has been suggested by a study of Cocchi [33] in which cow's milk was modified by removing half the casein and adding from 6 to 8 Gm. of lactalbumin per liter. Babies were nourished with this mixture for several months, the results showing that milk richer in lactalbumin produced regular growth. Cocchi concluded that it is not sufficient to satisfy merely the minimum protein needs of the infant but that it is necessary to consider also the minimum lactalbumin requirements. This apparent demand for albumin by certain

29. Brennemann, J.: Artificial Feeding of Infants, in Abt: Pediatrics, Philadelphia, W. B. Saunders Company, 1923, vol. 2. Southworth, T. S.: Substitute Infant Feeding, in Carr, W. L.: The Practice of Pediatrics, Philadelphia, Lea Bros. & Company, 1906, vol. 16, p. 125.

30. Griffith, J. P. C.: The Diseases of Infants and Children, Philadelphia, W. B. Saunders Company, 1919, vol. 1., pp. 48 and 99.

31. Rotch, T. M.: Pediatrics, ed. 3, Philadelphia, J. B. Lippincott Company, 1901.

32. Brennemann (footnote 29, first reference).

33. Cocchi, C.: Growth-Producing Amino-Acids and the Milk Feeding of the Baby, Boll. d. Soc. ital. di biol. sper. **4**:55, 1929; Chem. Abstr. **23**:3492, 1929.

infants has further been demonstrated by Langer and Schmal,[34] who fed minute amounts (0.2 Gm.) of albumin mixed with pumped breast milk to infants not thriving on breast milk, with resulting improvement in their condition.

We have found that the nonprotein nitrogen may be an important factor in the nutrition of infants, and one that is likely to be overlooked.[35] Courtney and Brown[36] in their studies on the nonprotein nitrogen of human milk called attention to the milk containing a very high nonprotein nitrogen that was taken by an infant with eczema. On the other hand, sensitizations to protein, shown by exclusively breast-fed infants affected with eczema, have suggested that minute amounts of foreign proteins in the milk may lead to disturbed conditions.[37]

In view of these observations, the nitrogen partition of the milk taken by the baby during the progression of nursing offers an interesting study relative to the baby's nutritional well-being. The data in table 5 indicate a definite increase of casein nitrogen in the last half of the milk expressed by subjects 6 and 9, while no definite trend is shown in that of subject 7. The noncasein protein nitrogen and the nonprotein nitrogen were shown to be sometimes higher and sometimes lower in the last portion of milk. The infant of subject 9 had a slight degree of eczema. It is interesting to note that the level for nonprotein nitrogen in the milk of this woman (study *a*), which was comparatively high, had decreased to a normal value two weeks later (study *b*) after the elimination from the mother's diet of some carbohydrates and certain protein foods, particularly eggs. During the interval of two weeks, the baby's eczema cleared up, owing either to the change in the milk brought about by the limited diet of the mother or to the shortening

34. Langer, H., and Schmal, S.: Ansatzsteierung nicht gedeihender Brustkinder durch kleine Eiweisszulagen, Ztschr. f. Kinderh. **39**:80, 1925.

35. Macy, I. G.; Outhouse, J.; Long, M. L.; Brown, M.; Hunscher, H., and Hoobler, B. R.: Observations upon the Composition of Blood and Milk of Women during the Different Stages of Lactation, J. Biol. Chem. **74**:31, 1927.

36. Courtney, A. M., and Brown, A.: The Protein and Non-Protein Fractions of Some Samples of Woman's Milk, Arch. Dis. Childhood **5**:36, 1930.

37. Talbot, F. B.: Eczema in Childhood, M. Clin. North America **1**:985, 1918. O'Keefe, E. S.: The Relation of Food to Infantile Eczema, Boston M. & S. J. **183**:569, 1920; Eczema in the Breast Fed Baby, and Protein Sensitization, ibid. **185**:194, 1921. Shannon, W. R.: Eczema in Breast Fed Infants as a Result of Sensitization to Foods in the Mothers Dietary, Am. J. Dis. Child. **23**:392 (May) 1922. Stuart, H. C.: The Excretion of Foreign Protein in Human Milk, Am. J. Dis. Child. **25**:135 (Feb.) 1923. Ratner, B.; Jackson, H. C., and Gruehl, H. L.: Transmission of Protein Hypersensitiveness from Mother to Offspring. III: The Rôle of Milk, J. Immunol. **14**:275, 1927. Donnally, H. H.: The Question of the Elimination of Foreign Protein (Egg White) in Woman's Milk, J. Immunol. **19**:15, 1930.

TABLE 5.—*Nitrogenous Constituents* *

Observations		Total Nitrogen			Casein Nitrogen			Protein Nitrogen Not Casein Nitrogen			Nonprotein Nitrogen			Amino-Acid Nitrogen		
Subject	Time	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference
7 (a)	10 a.m.	162	160	−1.2	55	47	−14	52	50	− 4	27.4	28.0	+ 2	4.3	3.9	− 9
	2 p.m.	160	161	+0.6	56	51	− 9	57	55	− 4	31.5	29.2	− 7	4.4	3.8	−14
	6 p.m.	163	169	+3.6	38	51	+25	80	69	−14	30.9	27.5	−11	4.7	4.4	− 6
	10 p.m.	147	153	+3.9	42	31	−26	61	74	+18	30.9	32.0	+ 3	4.5	4.0	− 9
	2 a.m.	151	156	+3.2	23	27	+15	82	80	− 2	30.4	30.4	0	3.6	3.2	−11
	6 a.m.	144	155	+7.0	40	32	−20	74	66	−11	30.4	28.0	− 8	3.3	3.1	− 6
7 (b)	10 a.m.	172	170	−1.2	47	50	+ 6	68	67	− 1	38.9	34.9	−10	4.9	4.2	−14
	2 p.m.	174	173	−0.6	55	53	− 4	69	70	+ 1	37.0	40.0	+ 8	3.7	4.7	+21
Range of percentage of difference			−1.2 to +7.0			−26 to +25			−14 to +18			−11 to +8			−14 to +21	
Average percentage of difference			+2			−3			−5			−3			−6	
6	10 a.m.	163	169	+3.6	59	65	+ 9	53	57	+ 7	32.0	32.0	0	4.4	5.0	+14.0
	2 p.m.	175	177	+1.1	65	66	+ 2	61	54	−11	33.2	34.3	+ 3	5.1	5.0	− 2.0
	6 p.m.	175	182	+3.8	56	63	+11	57	59	+ 3	34.9	37.8	+ 8	5.6	5.5	− 0.4
	10 p.m.	163	164	+0.5	56	61	+ 8	56	58	+ 3	33.2	29.2	−12	3.7	3.3	−11.0
	2 a.m.	169	175	+3.4	56	60	+ 7	58	55	− 5	34.4	35.6	+ 3	3.2	2.9	− 9.0
	6 a.m.	163	173	+5.8	54	60	+10	62	62	0	31.5	32.0	+ 2	2.8	2.8	0.0
Range of percentage of difference			+0.5 to +5.8			+2 to +11			−11 to +7			−12 to +8			−11 to +14	
Average percentage of difference			+3			+8			−5			+1			−2	
9 (a)	24 hr.	175	171	−2.7	60	68	+12	58	59	+ 2	44.8	45.2	+ 0.9	5.6	5.8	+ 3.0
9 (b)	24 hr.	165	166	+0.6	59	62	+ 5	83	57	−31	30.0	35.0	+14	6.0	6.1	+ 0.3
Median of percentage of differences			+2.1			+6.5			−1.5			+1.5			−6.0	

* The amounts are given in milligrams per hundred cubic centimeters of milk.

of the nursing period of the infant, or to both. This correlation between a high value for nonprotein nitrogen in the breast milk and eczema in the nursing infant corroborates the observations of Courtney and Brown.[36]

Controversy exists as to the cause of eczema, urticaria, asthma and other allergic symptoms that sometimes develop in infants fed exclusively on breast milk. This question has been renewed recently in an editorial [38] in which it is stated:

> The withdrawal of certain articles of food from the mother's diet often leads to cessation of these symptoms, eggs being the most common exciting agent. In a few cases skin tests have been made showing the infant's hypersensitivity to maternal milk, but attempts to identify the allergic excitant in this milk have thus far given negative results. . . . Pediatricians, therefore, have been forced to consider the possibility that the toxic agent is some individual metabolic or secretory abnormality in human milk, the egg diet being responsible for this maternal biochemical perversion.

Applying a new quantitative technic to human milk,[39] it has been shown that after an egg diet normal human whey often contains as much as one part in a million of apparently unaltered albumin, a sufficient quantity to account for allergic symptoms. On the other hand, the two cases of eczema in breast-fed infants existing concurrently with high nonprotein nitrogen of the milk, as previously cited, suggest the need for further detailed observation on metabolic or secretory abnormalities in nursing mothers.

The partition of the nonprotein nitrogen constituents in maternal milk may yield further information on abnormalities in the elaboration of milk. We shall present later a detailed study on this particular phase of the chemistry of breast milk. In this connection it is interesting to note that the amino-acid nitrogen usually tended to decrease between the first and end portions, as shown in table 5.

Individual differences are shown in these studies of the nitrogen partition of breast milk. The studies emphasize the possibility that variations between the first and last portions of milk expressed from the glands may vary among women, owing perhaps to differing types of structure of the mammary glands and other factors incorporated in the mechanism of the secretion itself, and also to extraneous influences. All such variations may have a nutritional as well as a clinical significance. Evidence of the nutritional and clinical importance of the ratio between the fat and the protein, the proportional amounts of casein and albumin, the total amount of nonprotein nitrogen and even

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38. Current Comment: Secretion of Alien Proteins in Human Milk, J. A. M. A. **95**:1593 (Nov. 22) 1930.

39. Footnote 38. Coca, A. F., and Grove, E. F.: Studies in Hypersensitiveness: A Study of the Atopic Reagins, J. Immunol. **10**:445, 1925.

its individual components serves to emphasize the importance of further detailed studies on the chemistry of breast milk and the corresponding nutritional status of the child.

Corresponding determinations of lactose at the beginning and end of nursing have given indefinite results, although it is generally stated that lactose is the most constant of all the constituents of milk. Some authors (table 3) have recorded no appreciable differences, or very indefinite changes, while others have found a definite diminution of lactose in the last portions of a nursing sample. Observations by Denis and Talbot [40] showed an average decrease of 15 per cent, while those recently published by Wittenberg [41] indicated that lactose, like fat, increases during the progression of nursing. Although determinations of lactose and the percentage of differences vary widely in samples of milk representative of the first and the last halves of nursing, as shown in table 4, the median (+ 0.2) would demonstrate a negligible change.

Lactose has been assigned a minor rôle in the causation of digestive disturbances in breast-fed babies. Gerstley and his co-workers [42] observed that an excess of lactose in either breast milk or cow's milk caused no immediate disturbance in the infant. Moreover, Brenneman [43] stated that although lactose occurs with remarkable constancy in breast milk, and in large amounts, it probably never causes any digestive disorders.

As would be anticipated from the preceding results of increased fat and protein in the milk at the end of nursing, the total solids likewise increased about 8 per cent during the progress of nursing (table 4) while the nonfat solids diminished about 5 per cent.

From a number of observations of the total ash in human milk, Barbier and Mascre [44] concluded that, although the concentration of mineral salts does not remain constant, the concentration does not appear to undergo fixed variations from the beginning to the end of a feeding period. Observations of other authors (table 3), based on occasional cases, yielded results that show differences in per cent between the two portions of milk that range from — 39 to + 17.

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40. Denis, W., and Talbot, F. B.: A Study of the Lactose, Fat, and Protein Contents of Women's Milk, Am. J. Dis. Child. **18**:93 (Aug.) 1919.

41. Wittenberg, A.: Die Milchzuckerbestimmung in kleinen Mengen von Frauenmilch, Monatschr. f. Kinderh. **32**:510, 1926.

42. Gerstley, J. R.; Wang, C. C.; Boyden, R. E., and Wood, A. A.: The Influence of Feeding on Certain Acids in the Feces of Infants: II. The Effect of an Excess of Lactose in Breast Milk in Modified Cow's Milk, with Observation on a Parenteral Infection, Am. J. Dis. Child. **36**:289 (Aug.) 1928.

43. Brennemann (footnote 29, first reference).

44. Barbier, H., and Mascre: Note sur les variations des sels minéraux du lait de femme aux différentes heures de la journée, Bull. Soc. de pédiat. de Paris **13**:260, 1911.

Differences between persons are illustrated in the analysis of the total ash and some mineral constituents in the milk of our subjects. In table 6 the percentage of differences between corresponding analyses of the total ash are shown. In the milk of subject 7 the percentage of differences of total ash varies between — 5 and + 4; in that of subject 6 there is an increase in the last half, and in that of subject 9 the amount of ash remains about constant.

From the evidence of numerous experimental studies [45] on the relative amounts of calcium, phosphorus, other salts and fat in human milk, it would seem that these constituents must be present in suitable combinations and proportions for optimal mineral utilization by the infant. Sisson and Denis [46] suggested that derangements of salt in the milk may explain many nutritional disorders among infants. This idea is substantiated by recent studies of Courtney and Brown,[47] in which a group of infants showing unfavorable nutritional symptoms took milks that exhibited abnormal values for two or more salts. Experiments of Bosworth, Bowditch and Giblin [48] on the effects of excess calcium in the utilization of fat by infants led to the following statement:

> The influence of salts of milk both on account of their nutritive value and the influence they have on other food components of milk, demand more consideration than they have received in infant feeding. The influence of salts on the mechanical condition of milk after it reaches the stomach, on the digestibility of the food components after digestion, and the utilization of the vitamins of the milk are pertinent questions.

In view of the foregoing considerations, it is noteworthy to observe the variations of individual ash components in the milk at the beginning and end of nursing as reported in the few references recorded in the

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45. Bosworth, A. W.: Studies of Infant Feeding: XIII. The Caseins of Cow's Milk and Human Milk in their Relation to Infant Feeding. The Action of Rennin on Casein, Am. J. Dis. Child. **22**:193 (Aug.) 1921. Wang, C. C.; Witt, O. B., and Felcher, A. R.: A Comparison of the Metabolism of Some Mineral Constituents of Cow's Milk and of Breast Milk in the Same Infant, ibid. **27**:352 (April) 1924.

46. Sisson, W. R., and Denis, W.: Observations on the Salt Content of Human Milk, J. A. M. A. **75**:601 (Aug. 28) 1920.

47. Courtney, A. M., and Brown, A.: Salt Content of Woman's Milk in Some Cases in Which Its Use Was Not Beneficial, Arch. Dis. Childhood **5**:28, 1930.

48. Bosworth, A. W.; Bowditch, H. I., and Giblin, L. A.: Studies of Infant Feeding. X: The Digestion and Absorption of Fats, I, Calcium in Its Relation to the Absorption of Fatty Acids, Am. J. Dis. Child. **15**:397 (June) 1918. Bowditch, H. I., and Bosworth, A. W.: Studies of Infant Feeding. IX: The Availability of Dicalcium Phosphate When Present as a Constituent of Infant's Food, Boston M. & S. J. **177**:864, 1917.

TABLE 6.—*Mineral Constituents* *

Observations		Ash			Chlorine			Calcium			Phosphorus			Ratio—Ca : P		
Subject	Time	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference
7 (a)	10 a.m.	203	201	+1.0	43.9	45.0	+ 4.2	34.3	34.4	+ 0.3	15.7	15.5	— 1.3	2.18	2.22	+ 1.8
	2 p.m.	212	216	+1.9	40.3	40.3	0	35.0	34.8	— 0.6	15.6	15.5	— 0.6	2.25	2.24	— 0.5
	6 p.m.	213	203	—4.7	39.3	41.8	+ 6.0	31.9	31.5	— 1.3	15.5	15.7	+ 1.3	2.06	2.01	— 2.4
	10 p.m.	211	205	—2.8	43.9	43.5	— 0.9	32.5	33.3	+ 2.4	14.5	14.1	— 2.8	2.25	2.36	+ 4.7
	2 a.m.	208	198	—4.8	40.0	40.7	+ 1.7	30.6	32.0	+ 4.4	13.5	14.5	+ 6.8	2.27	2.21	— 2.6
	6 a.m.	206	201	—2.4	46.0	45.7	— 0.7	29.9	30.8	+ 2.9	13.4	15.2	+11.8	2.23	2.02	— 9.4
7 (b)	10 a.m.	198	195	—1.5	....	....	.....	32.1	31.8	— 0.9	14.4	15.1	+ 4.6	2.23	2.10	— 5.8
	2 p.m.	203	211	+3.8	....	....	.....	37.9	33.5	—11.6	15.2	16.9	+10.0	2.49	1.98	—12.6
Range of percentage of difference			—4.8 to +3.8			—0.9 to +6.0			—11.6 to +4.4			—2.8 to +11.8			—12.6 to +4.7	
Average percentage of difference			—1.4			+2.1			—0.6			+3.7			—3.4	
6	10 a.m.	204	215	+5.1	38.0	38.0	0	31.7	32.0	+ 0.9	12.1	13.5	+10.4	2.61	2.37	— 9.2
	2 p.m.	236	240	+1.7	33.7	35.5	+ 5.1	36.8	38.6	+ 4.7	13.0	14.2	+ 8.5	2.84	2.72	— 4.2
	6 p.m.	224	233	+3.9	36.2	37.3	+ 2.9	35.5	36.5	+ 2.7	13.6	14.9	+ 8.7	2.61	2.45	— 6.1
	10 p.m.	213	223	+4.5	31.9	32.6	+ 2.1	34.9	35.1	+ 0.6	13.2	14.7	+10.2	2.64	2.38	— 9.8
	2 a.m.	213	231	+7.8	37.6	34.0	— 6.4	34.5	34.6	+ 0.3	13.2	14.7	+10.2	2.62	2.35	—10.3
	6 a.m.	208	217	+4.1	38.4	35.8	— 6.8	33.3	32.3	— 3.0	12.4	13.6	+ 8.8	2.69	2.37	—11.9
Range of percentage of difference			+1.7 to +7.8			—6.8 to +5.1			—3.0 to +4.7			+8.5 to +10.4			—11.9 to —4.2	
Average percentage of difference			+4.5			—0.6			+1.0			+7.1			—8.7	
9 (a)	24 hr.	197	197	0	....	....	.....	37.7	37.4	— 0.8	12.9	12.5	— 3.1	2.92	2.99	+ 2
9 (b)	24 hr.	199	198	—0.5	....	....	.....	37.3	37.6	+ 0.7	13.3	13.4	+ 0.4	2.80	2.80	0
Median of percentage of differences			+0.8			+0.9			+0.4			+7.7				

* The amounts are given in milligrams per hundred cubic centimeters of milk.

literature. St. Engel [49] showed variations in several mineral constituents of human milk at the beginning and close of nursing. These are presented in table 7.

The analyses of Sisson [50] on the chloride content of the samples of milk of normal women obtained at the beginning and the end of the nursing period showed considerable but indefinite fluctuations. Corroborating these observations, the chloride content of the milk of

TABLE 7.—*Statistics on Mineral Constituents of Human Milk (St. Engel*)*

Constituent	Begin	End	Constituent	Begin	End
K_2O	100.8	63.4	Fe_2O_3	0.22	0.12
Na_2O	44.8	17.6	P_2O_5	32.10	28.88
CaO	37.6	38.1	SO_3	9.6	7.2
MgO	5.4	5.2	Cl	71.7	34.2

* The amounts are given in milligrams per hundred cubic centimeters of milk.

TABLE 8.—*Statistics on Calcium in Milk (Hunaeus)*

Beginning	End	Percentage of Difference
0.0465	0.0471	+1.2
0.0450	0.0446	—0.9
0.0406	0.0389	—4.1
0.0460	0.0424	—7.8
0.0381	0.0345	—9.4
0.0460	0.0450	—2.1

TABLE 9.—*Calcium Oxide Before and After Nursing (Stransky)*

Case	Before Nursing, per Cent	During Nursing, per Cent	After Nursing, per Cent	Percentage of Difference
A	0.66	0.35	0.28	—58
B	0.98	0.48	0.28	—71
C	0.97	0.35	0.29	—70
D	0.84	0.38	0.30	—60

our subjects 6 and 7 changed irregularly with the progression of nursing, as shown by the median — 0.9 in table 6.

Hunaeus [51] found that in five or six cases which he studied, the calcium oxide in the milk at the end of the nursing period was definitely lower than at the beginning (table 8).

49. St. Engel: Die Frauenmilch, in Sommerfeld: Handbuch der Milchkunde, Wiesbaden, 1909; cited by Levine, V. E.: The Differences Between Cow's Milk and Woman's Milk, Arch. Pediat. **43**:353, 1926.

50. Sisson, W. R., and Denis, W.: Studies on the Inorganic Constituents of Milk, Am. J. Dis. Child. **21**:389 (April) 1921.

51. Hunaeus: Ueber den Kalkgehalt der Frauenmilch, Biochem. Ztschr. **22**: 442, 1909.

Recent investigations by Stransky [52] demonstrated an even greater magnitude of diminution of the calcium oxide in the last portion of the milk, as shown by table 9.

In contrast to results published by other workers on samples of milk collected before and after the baby nursed, the calcium studies on the milk of the women of this investigation did not show any definite trend in the milk of the first and last halves of nursing (table 6).

Little attention has been given to the phosphorus of human milk collected at the beginning and end of the nursing, though it would seem of considerable importance in the nutritional well-being of the child. In the present study, corresponding determinations of phosphorus illustrated a general tendency toward augmentation as nursing progressed (table 6).

TABLE 10.—*Ratios of Certain Constituents*

Observation		Fat : Protein Ratio			Fat (Gm.) : Phosphorus (Mg.) Ratio			Cl (Mg.) × 100 / Lactose		
Subject	Time	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference	First Half	Last Half	Percentage of Difference
7 (a)	10 a.m.	4.0	5.5	+27	0.26	0.36	+28	0.63	0.62	— 2
	2 p.m.	3.8	4.9	+23	0.25	0.32	+22	0.56	0.57	— 2
	6 p.m.	3.8	5.6	+32	0.25	0.39	+36	0.57	0.61	+ 7
	10 p.m.	3.7	6.3	+41	0.24	0.44	+45	0.61	0.60	— 2
	2 a.m.	2.9	3.8	+24	0.21	0.26	+19	0.54	0.57	+ 5
		1.5	1.9	+21	0.10	0.12	+17	0.64	0.63	— 2
7 (b)	10 a.m.	3.1	4.1	±24	0.23	0.29	+21			
	2 p.m.	2.9	3.8	±24	0.21	0.25	+16			
6	10 a.m.	5.6	7.7	+27	0.49	0.61	+20	0.53	0.53	0
	2 p.m.	4.5	5.6	+20	0.38	0.45	+16	0.47	0.48	+ 2
	6 p.m.	3.0	5.1	+41	0.25	0.39	+36	0.51	0.53	+ 4
	10 p.m.	3.7	4.9	+24	0.29	0.35	+17	0.44	0.47	+ 6
	2 a.m.	3.4	4.2	+19	0.28	0.31	+13	0.52	0.48	— 4
	6 a.m.	3.6	4.3	+16	0.31	0.34	+ 9	0.53	0.50	+ 6
Median.....				±24			±20			±1

Table 10 shows the ratios of some of the constituents in the milk at the first and the last halves of nursing. These are significant in connection with experiments on utilization of the components of the milk by babies when one or several of the constituents are varied considerably, but it is beyond the scope of this paper to discuss in detail the nutritional and clinical importance of these interrelationships. It was previously suggested [15] that constant ratios between two constituents throughout a nursing period would perhaps indicate at least a close association in the mechanism of their secretion. The ratio between the fat and the protein was consistently higher in the last half of the nursing period, indicating that though both these constituents may increase, the fat seems to increase much more in proportion. The same situation

52. Stransky, E.: Zur Kenntnis des Kalkgehaltes der Frauenmilch, Ztschr. f. Kinderh. **40**:671, 1926.

is observed in the ratio between the fat and the phosphorus, which shows that these constituents did not increase at the same rate. The behavior of the phosphorus is likewise shown in table 6 in the ratio of calcium to phosphorus, which shows a definite decrease in the last half and may demonstrate that calcium and phosphorus are not secreted at the same rate throughout nursing. Such evidences of varying rates of secretion may lend support to the hypothesis that the secretion of fat is controlled by an entirely separate mechanism.[13]

The constancy of the ratio of chlorine to lactose and its significance in the differentiation of human milk and cow's milk have been demonstrated recently.[53] Calculation of the chlorine-lactose quotient in the milk of the first and last halves of the nursing period illustrates in general such a constancy, which gives further evidence that these two components are closely associated and are perhaps secreted alike. Physicochemical studies by Jackson and Rothera [54] on depression of the freezing point and electrical conductivity of the first and last portions of human milk at the time of nursing led them to believe that differences in these constants bear out the assumption that the first and last portions are secreted under different conditions.

CONCLUSIONS

Renewed interest has been focused on the chemistry of breast milk as it relates to the nutritional well-being of the baby at the breast. Under standardized methods of collection and analysis, gross chemical changes in breast milk representative of the first and last halves of nursing have been observed. These changes were not influenced by the presence and condition of the baby at the breast. They are indicative of nutritional and clinical significance. During the progression of nursing, the fat, protein, casein-nitrogen, total solids and phosphorus tend to increase. It has been pointed out that definite ratios between various constituents in the milk are of importance. These ratios, with the progression of nursing, may approach abnormal values in some cases. Furthermore, the inconstancy of ratios between certain constituents lends support to the indications that certain components are controlled by different secretory mechanisms.

53. Keller, A. (Munich), and Mai, H.: Untersuchungen über die Zusammensetzung der Milch, Schweiz. med. Wchnschr. **60**:487, 1930.

54. Jackson, L. C., and Rothera, A. C. H.: Milk: Its Milk Sugar, Conductivity and Depression of Freezing Point, Biochem. J. **8**:1, 1914.

Case Reports

ACUTE MASSIVE ATELECTASIS AND ASTHMA*

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Acute massive atelectasis of the lung was accurately described in 1890 by Pasteur.[1] The studies of Bradford [2] in connection with war wounds were largely responsible for the renewal of interest in this subject. Numerous and important contributions have appeared in the literature in the last five years.

Acute massive atelectasis of the lung usually occurs after abdominal operations or thoracic traumas. The nonsurgical and nontraumatic occurrence of acute massive atelectasis of the lung as a complication has been described in such diseases and conditions as diphtheria,[1] acute meningitis,[3] poliomyelitis,[4] pneumonia,[5] acute [6] and chronic [7] suppurations of the lung, carcinoma,[8] pulmonary tuberculosis,[9] aneurysm of

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* From the Children's Asthma Clinic, Mount Sinai Hospital, Dr. Bela Schick, Director of the Department of Pediatrics.

1. Pasteur, W.: Respiratory Paralysis After Diphtheria as a Cause of Pulmonary Complications, Am. J. M. Sc. **100**:242, 1890.

2. Bradford, J. R.: The Bradshaw Lecture on Massive Collapse of the Lung, Lancet **2**:1351 (Nov. 7) 1908; The Oxford Medicine, New York, Oxford University Press, 1920, vol. 2, p. 127.

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9. Packard, E. N.: Massive Collapse (Atelectasis) Associated with Pulmonary Tuberculosis and Tumor, Am. Rev. Tuberc. **18**:7 (July) 1928.

the aorta,[10] intrabronchial tumor,[11] foreign body in the bronchus [12] and hyperplasia of the thymus.[13]

ETIOLOGY

The etiology of acute massive atelectasis of the lung is obscure. Some evidence is available as to the pathogenesis. In 1879, Lichtheim [14] showed experimentally that blocking of the bronchus and ligation of the vessels to the obstructed lung failed to result in atelectasis, indicating that massive atelectasis was the result of absorption of the air in the entrapped alveolae by the circulating blood. These observations were further stressed by Coryllos and Birnbaum,[15] and Jackson and his associates [16] who have shown that bronchial obstruction, irrespective of the type or character of the obstructing agent, remains the only definitely proved constant factor. The question of atelectasis occurring during the course of acute and chronic diseases, entirely independent of a history of trauma or operation, involves interesting controversial discussion. Probably the obstructing agent inducing atelectasis is only the exciting

10. Ylvisaker, R.: Massive Collapse of the Lung Due to Aortic Aneurysm; Report of two Cases, Minnesota Med. **11**:483 (July) 1928. Francis, B. F.: Small Aneursym Pressing on Left Bronchus with Complete Collapse of the Lung, J. Missouri M. A. **26**:134 (March) 1929.

11. Ashbury, H. E.: Recurrent Massive Collapse of the Lung Due to Benign Intrabronchial Tumor, Am. J. Roentgenol. **21**:452 (May) 1929.

12. McCrae, T.: The Physical Signs of Foreign Bodies in the Bronchi, Am. J. M. Sc. **159**:313 (March) 1920. Manges, W. F.: Atelectasis as a Roentgen-Ray Sign of Foreign Bodies in the Air Passages, Am. J. Roentgenol. **11**:517 (June) 1924. Jackson, C.: Pulmonary Collapse Due to Foreign Bodies in the Bronchi, Tr. Am. Lung A. **43**:39, 1925.

13. Costello, J. P.: Thymic Enlargement as Cause of Atelectasis, M. Clin. North America **11**:583 (Sept.) 1927. Jacobson, H. J.: Thymic Atelectasis; Case Report, Memphis M. J. **5**:107 (May) 1928. Slot, G. M.: Two Cases of Familial Enlargement of the Thymus Associated with Collapse of the Lung, Lancet **2**:652 (Sept. 29) 1928.

14. Lichtheim, L.: Versuche über Lungenatelektase, Arch. f. exper. Path. u. Pharmakol. **10**:54, 1879.

15. Coryllos, P. N., and Birnbaum, C. L.: Obstructive Massive Atelectasis of the Lung, Arch. Surg. **16**:501 (Feb.) 1928.

16. Jackson, C., and Lee, W. E.: Acute Massive Collapse of the Lung; Discussion of its Mechanism and of its Relation to Foreign Bodies in Bronchi and Postoperative Complications, Ann. Surg. **82**:364 (Sept.) 1925. Tucker, G.: Recent Developments in Peroral Endoscopy; Oesophagoscopy and Bronchoscopy for Disease, Surg. Gynec. Obst. **42**:743 (June) 1926. Clerf, L. H.: Postoperative Massive Collapse of the Lung, Report of Bronchoscopic Observations, Ann. Surg. **85**:54 (Jan.) 1927. Lee, W. E.; Ravdin, I. S.; Tucker, G., and Pendergrass, E. P.: Studies on Experimental Pulmonary Atelectasis; Production of Atelectasis, Ann. Surg. **88**:15 (July) 1928.

or secondary factor, and the underlying or primary etiologic factor becomes a subject for theoretical consideration. The theoretical explanations of the fundamental causative factors range from immobility of the patient or a tendency on the part of the patient to splint respiratory movements in all operations especially those on the thorax and abdomen,[2] to bronchial spasm due to reflex irritation in other parts of the body.[17]

Hirschboeck [18] reported the case of a woman, 44 years of age, in whom massive atelectasis of the left lung developed on the day following cholecystectomy. Five days later pneumonia on the contralateral, right side developed. The patient died on the seventh day. Necropsy disclosed a bronchial obstruction on the left side due to pressure from an aneurysm of the arch of the aorta, which undoubtedly antedated the operation for some time. Interestingly enough, the operation had induced an attack of massive atelectasis of the lung within forty-eight hours. The most plausible explanation for this occurrence is found in either of the foregoing theories. In this case bronchial obstruction seems to be the exciting or secondary factor in causing the atelectasis.

Elliot and Dingley [19] and others, assuming the condition to be primarily due to obstruction of the bronchial tube by secretion, found no such obstruction at necropsy.

ATELECTASIS IN ASTHMA

From the reports in the literature, acute massive atelectasis of the lungs as a complication of a common ailment like bronchial asthma appears to be a relatively rare condition. All of the cases reported were in adults, as follows.

Birkhart [20] reported a case of chronic asthma in a woman, aged 37, who died; at necropsy, a plug in the bronchus with areas of partial atelectasis and emphysema was found.

Kamchorn and Ellis [21] found at necropsy also a partial atelectasis of the left lung and of the lower lobe of the right lung in a man, aged 52, who had suffered from asthma since childhood.

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17. Scott, W. J. M.: Postoperative Massive Collapse of the Lung, Arch. Surg. **10**:73 (Jan.) 1925. Scott, W. J. M., and Joelson, J. J.: Post-Operative Massive Atelectasis: I. Influence of Posture, Arch. Surg. **15**:855 (Dec.) 1927. Sante, L. R.: Massive Collapse of the Lung, Radiology **8**:1 (Jan.) 1927.

18. Hirschboeck, E. J.: Massive Collapse of the Lung, with Report of Necropsy, Minnesota Med. **11**:135 (March) 1928.

19. Elliot, T. R., and Dingley, L. A.: Massive Collapse of the Lung Following Abdominal Operation, Lancet **1**:1305 (May 9) 1914.

20. Birkhart, J. B.: On Bronchial Asthma: Its Pathology and Treatment, London, J. A. Churchill, 1889.

21. Kamchorn, N., and Ellis, A. G.: The Pathology of Bronchial Asthma, Am. J. M. Sc. **161**:525 (April) 1921.

Kessel and Hyman [22] reported a case of atelectasis of the lower lobe of the left lung, confirmed by bronchoscopy, in a woman aged 52, who had attacks of asthma for five years. Under bronchoscopic treatment, the patient made an uneventful recovery. In six months the roentgenogram of the lungs was negative, and she was completely relieved from asthma.

Sante [23] reported a case in a boy, aged 19, who had suffered from attacks of asthma for seven years, and in whom massive atelectasis of the left lung with pneumothorax developed during an attack of asthma. The patient recovered.

Clarke [24] reported the cases of two patients with a history of bronchial asthma of many years' standing. Roentgenoscopy of the chest of the first patient showed complete atelectasis of the lower lobe of the right lung. A few days later bronchoscopy did not reveal any bronchial obstruction, while in the roentgenogram the lung still showed atelectatic collapse. There was complete recovery. The other patient died of severe dyspnea and cyanosis twelve hours after the onset of the symptoms. At necropsy, both lower lobes were collapsed.

In a study on the incidence of allergic diseases in ten patients in whom postoperative atelectasis had developed, Wilmer, Cobe and Lee [25] found that all showed previous evidences of allergic sensitization. The age of the patients ranged from 12 to 59 years. The patient, 12 years of age, had hay-fever and was the only child in the series. Of the remaining nine patients, one had urticaria, two vasomotor rhinitis, one chronic eczema, one hay-fever, one asthma and three gave positive reactions to skin tests of various allergens without symptoms of allergy. These observations hint at the possibility that allergic persons are predisposed to the postoperative complications of acute massive atelectasis of the lung.

A roentgenoscopic study [26] of over 500 children with bronchial asthma including almost 1,000 roentgenograms failed to reveal a single case of massive atelectasis, partial or complete, as a nonsurgical complication of asthma.

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22. Kessel, L., and Hyman, H. T.: Some Unusual and Atypical Intrathoracic Conditions, Am. J. M. Sc. **175**:511 (April) 1928.

23. Sante, L. R.: Massive (Atelectasis) Collapse of the Lung, With Report of a Case Showing Associated Spontaneous Pneumothorax, Am. J. Roentgenol. **20**:213 (Sept.) 1928.

24. Clarke, J. A., Jr.: Pulmonary Atelectasis Complicating Bronchial Asthma, J. Allergy **1**:81 (Nov.) 1929.

25. Wilmer, H. B., Cobe, H. M., and Lee, W. E.: Incidence of Allergy and Asthma in Group Developing Post-Operative Atelectasis, Ann. Surg. **91**:651 (May) 1930.

26. Peshkin, M. M.: Asthma in Children: V. Roentgenoscopy of the Chest and the Tuberculin Skin Reaction, to be published.

The following case of a boy is believed to be one of rare interest because the onset of asthma was initiated by a massive atelectasis of the lung (nontuberculous and without surgical indications).

REPORT OF A CASE

History.—R. L., a boy, aged 9, was referred to Mount Sinai Hospital on June 22, 1928, with a diagnosis of pleurisy with effusion, and a history of apparent well-being until one week prior to admission, when in the act of falling, he was struck on the chest by the handle of his bicycle. Following this accident, the patient did not complain of pain in the chest nor was there evidence of external injury. Two days later he had a cough without wheezing and a slight elevation in temperature from 100 to 101 F., which was maintained up to the time of admission to the hospital. Three days before admission, the child was examined at another institution where the mother was informed that he was well. On the way home he had a coughing spell followed by a severe pain in the left side of the chest with dyspnea.

Infancy had been normal except for a history of recurrent "abdominal colic" and frequent vomiting. Measles and pertussis occurred when the patient was 5 years old and varicella when he was 8½. Tonsillectomy and adenoidectomy had been performed.

The mother had had asthma for many years and a maternal uncle, hay-fever. The family history was otherwise negative to allergy.

Examination.—When seen at the hospital for the first time, the child appeared to be in great distress. He was markedly dyspneic and cyanosed. He had a persistent cough and complained of severe pain in the left side of the chest. He was perspiring profusely. The temperature was 100.4 F.; the rates of respiration and pulse were 40 and 140, respectively. The systolic blood pressure was 100, and the diastolic 56. Fifteen minutes after the injection of a sedative, the pulse rate was lowered and of improved quality, the dyspnea was less distressing, and the pain in the chest was controlled.

Physical examination of the chest showed the right side to be moving freely and the breath sounds to be normal. The motility of the left side of the chest was definitely diminished, and it appeared smaller than the right. On percussion of the left side of the chest anteriorly, there was flatness extending from the level of the third rib to the base of the lung; posteriorly, the flatness extended from the spine of the scapula down to the base of the lung. The breath sounds ranged from diminished to absent over the area of flatness with signs of compressed breathing above this area. The heart was displaced to the left.

On the following day the temperature was 101.6 F., the pulse rate 130 and that of respiration 60 per minute. Percussion of the chest gave a hyperresonant note on the right side and at the apex of the left lung. The breath sounds were entirely absent throughout the left side of the chest, except for the apex where musical râles were heard. Musical râles were present throughout the right side of the chest.

Course.—On June 25, the patient was still markedly dyspneic but less cyanosed. The physical signs of asthma were prominent, to be heard now in both lungs, and persisted in spite of injections of epinephrine hydrochloride (1:1,000 solution), morphine and atropine sulphate. On the following day, there was moderate dyspnea with slight cyanosis, and asthmatic râles were heard only on auscultation.

On June 27, the boy was comfortable and spoke for the first time without much effort. Respirations still ranged between 40 and 50 per minute, but breathing was not labored. The temperature was 102 F., and the pulse rate 125. On this day, bronchoscopy [27] showed the left main bronchus and all the branches to be filled with a solid mass. Thick, stringy material extended into the finer bronchioles. The plug was removed, and the bronchus was irrigated with compound solution of iodine, the procedure being carried out with comparative ease. Several hours later, examintion of the chest showed that the right side, which formerly had been prominent, was now about the same size as the left. The percussion note over the right side was less emphysematous. The left side of the chest, posteriorly, seemed normal on percussion, while the note anteriorly was of tympanitic quality. Auscultation revealed all asthmatic râles to be absent. The breath sounds could be heard on both sides. The left border of the heart appeared to be in normal position.

On June 28, both halves of the thorax were the same in all diameters. Sibilant and sonorous râles were heard throughout the lungs. Toward evening the temperature rose to 103 F., the respiration rate was 48 and the pulse rate 130. Dyspnea and cyanosis were moderately increased. Physical examination suggested the development of acute bronchopneumonia. The boy appeared better on the following day. Examination of the chest showed only signs of bronchitis and asthmatic breathing. On July 4, he was afebrile for the first time. The lungs showed only a few, scattered, sibilant and sonorous râles.

The Pirquet, Mantoux and Schick tests gave negative results. The blood count on June 23 was normal except for a slight leukocytosis. The urine was normal. The protein skin tests gave negative results. The bronchial plug contained degeneration leukocytes of all types. Culture of the mucus obtained from the bronchus during bronchoscopy showed *Streptococcus hemolyticus*. Cultures taken from the nose and throat were negative for diphtheria bacilli.

Roentgenoscopy and fluoroscopy [28] of the chest on June 23 (fig. 1) showed a marked decrease in the volume of the left side of the chest, with a displacement of the heart and mediastinum to that side. The entire pulmonary field was obscured by a dense homogeneous shadow. The left side of the diaphragm was elevated. On June 25, there was an almost complete clearing of the left pulmonary field. The heart was still displaced to the left. On the following day there was complete clearing of the left pulmonary field with displacement of the heart to the left side. On June 27, the heart was in the normal position (fig. 2), and there was no abnormality in the lungs. Fluoroscopy showed both sides of the diaphragm to be moving normally, whereas on the previous day the left side of the diaphragm was practically immobile. On June 29, a roentgenogram of the chest showed a dense infiltration extending outward from the root of the left lung as far as the midclavicular line. There were also a few scattered infiltrations in the left lower and right upper lobes. These infiltrations had the appearance of a pneumonic process. On July 4, both lungs were well aerated, except for a small area at the root of the left lung. On July 11, there was a small area of infiltration at the root of the left lung, which was apparently undergoing absorption. Roentgenograms of the chest taken on Sept. 5, Nov. 5 and Dec. 5, 1928, and on Oct. 30 1930, showed no abnormality of the heart and lungs.

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27. The bronchoscopic examinations were performed by the staff of the Nose and Throat Department, Dr. S. Yankauer, director.

28. The roentgenoscopic and fluoroscopic examinations were performed by the Department of Roentgenology, Dr. L. Jackes, director.

The child was discharged from the hospital on July 19, 1928, and was sent to a convalescent home where he remained three weeks. During this period he was well except for slight cough and occasional wheezing spells.

Second Admission.—He was seen at the children's asthma clinic on the following September 4. The Pirquet and Mantoux tests were repeated with negative results. On September 20, he contracted an infection of the upper respiratory tract associated with slight elevation in temperature, cough and wheezing. The

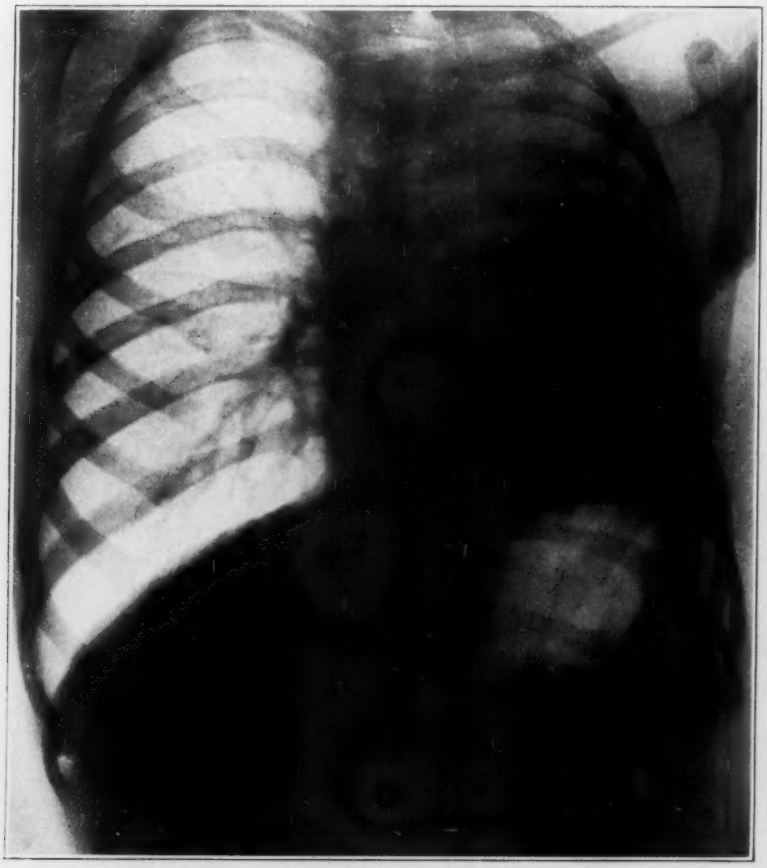

Fig. 1.—Marked decrease in the volume of the left side of the chest with emphysema of the right lung, June 23, 1928. The left pulmonary field is obscured by a dense homogeneous shadow. The left side of the diaphragm is elevated and the heart and mediastinum pushed over to the same side.

low grade temperature persisted, and on October 30 he was readmitted to the hospital. On November 8, bronchoscopy showed the bifurcation to be markedly thickened and a small amount of mucus in the lower lobe of the left lung. Asthmatic râles were heard before and after bronchoscopy. The results of the Pirquet and Mantoux tests (up to 5 mg. tuberculin) were again negative. Examinations of the blood and urine gave negative results, except that the polymorphonuclear

leukocytes in the blood were 12,400. The patient was well and was discharged from the hospital on November 15.

Course.—Several weeks later the boy returned to the clinic with a history of spells of recurrent wheezing and on one occasion a severe attack of asthma. In March, 1929, nonsyphilitic therapy was instituted and continued over a period of three months, with marked relief from asthma. During the summer he lived at a seashore resort, with continued freedom from asthma up to Nov. 6, 1930, when he was last seen.

GENERAL CONSIDERATIONS

On the basis of this case and of reports in the literature, acute massive atelectasis of the lung is usually found to occur from one to

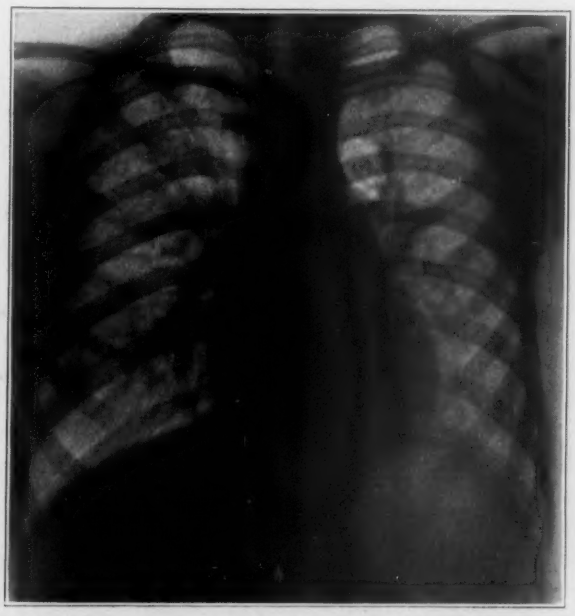

Fig. 2.—The heart and the mediastinum in the normal position, June 27, 1928.

four days after an operation or injury or as a complication during the course of various acute and chronic diseases. The onset is usually sudden, and cyanosis, dyspnea and pain in the chest are the common symptoms. The severity of these symptoms depends on whether there is an involvement of a whole lung, one lobe or a portion of a lobe, or even both lungs.[29] The lower lobes are more commonly involved than the upper. The patient tends to lie on the affected side. There are usually a rise in temperature and an increase in pulse rate.

29. Bergamini, H., and Shepard, L. A.: Bilateral Atelectasis (Massive Collapse) of the Lung, Ann. Surg. **86**:35 (July) 1927.

The affected side of the chest is definitely retracted and immobile. On percussion, there is dulness and flatness over the involved area. Breath sounds, vocal resonance and vocal fremitus may be absent, decreased or increased. The frequent variation of signs is usually due to a change in the degree of airlessness of the lung. The signs in the lungs with extensive involvement may be those of consolidation even to the extent of bronchial breathing. Râles and adventitious sounds are often absent in the early course of the most marked cases. Adventitious sounds may be a prominent sign in the later stages when the lung is reexpanding, followed by cough with expectoration of sputum. Inflammatory complications, such as bronchitis, pleurisy or bronchopneumonia, commonly develop in the atelectatic lung.

In a normal person, pulmonary expansion and the normal relationship of the thoracic organs are maintained by atmospheric pressure exerted intrapulmonarily via the trachea, and by the negative pressure equally exerted in both pleura. A release of this negative pressure by the entrance of air or fluid into the pleura is followed by a true collapse of the lung with the negative pressure falling or becoming positive. The term "collapse" when applied to massive atelectasis is misleading because separation of the lung from the wall of the chest does not take place. The suction or negative pressure normally existing in the pleura is markedly increased [30] and the heart, mediastinum and diaphragm are drawn toward the affected side as if it were a vacuum. This marked increase in intrapleural negative pressure draws the obstructing medium more firmly and deeply into the bronchus.

The cardinal signs in massive atelectasis of a lung are displacement of the heart and mediastinum to the affected side, with retraction and immobility of the wall of the chest, which is diminished in all its measurements, and the characteristic roentgenoscopic and fluoroscopic observations of immobility and raised dome of the diaphragm, together with a diffuse, dense pulmonary shadow and cardiac displacement on the affected side. These striking signs sharply differentiate cases of massive atelectasis from other pulmonary pleural lesions.

PROGNOSIS AND TREATMENT

The prognosis of massive atelectasis is favorable when only one lung is involved. Complete clearing of the affected lung, not treated bronchoscopically, takes place on an average in twenty-one days.[18]

When available, bronchoscopy [16] with aspiration or extraction of the obstructing agent, is regarded as the ideal method of treatment. It

30. Farris, H. A.: Atelectasis of Lung, Canad. M. A. J. **15**:808 (Aug.) 1925. Solem, G. O.: Massive Collapse of the Lung, M. Clin. North America **11**:105 (July) 1927. Elkins, D. C.: Intrapleural Pressure in Postoperative Atelectasis, Ann. Surg. **86**:885 (Dec.) 1927. Habliston, C. C.: Intrapleural Pressure in Massive Collapse of the Lung, Am. J. M. Sc. **176**:830 (Dec.) 1928.

is performed to hasten recovery, which usually occurs within three days, and also to avoid further septic complications, such as pneumonia or abscess. However, it has been found that if the patient is rolled to his good side and made to cough, his lungs almost immediately become reinflated.[31] This observation suggests the immediate usefulness of this method of treatment and does not carry any risk to the patient. Hyperventilation by carbon dioxide has also been advocated.[32] Deflation is prone to recur after each of the methods of treatment, especially with the hyperventilation by carbon dioxide. Artificial, partial pneumothorax, which reduces the intrapleural negative pressure to within normal limits, is employed by some authors,[30] because this treatment affords immediate symptomatic relief. Finally, sudden spontaneous improvement occurs not infrequently in the absence of any special treatment.

COMMENT

Diseases of childhood, such as pertussis, measles, scarlet fever and pneumonia as well as operations for the removal of tonsils and adenoids, collectively have been shown to initiate the onset of asthma in 38 per cent of a series of asthmatic children.[33] In another 3 per cent of the cases,[34] the onset of asthma was initiated by pathologic conditions causing a partial stenosis somewhere along the tracheobronchial tree. This phase of asthma has been referred to as para-asthma,[35] and the

31. Sante, L. R.: Massive (Atelectatic) Collapse of the Lung, with Especial Reference to Treatment, J. A. M. A. **88**:1539 (May 14) 1927.

32. Scott, W. J. M., and Cutler, E. C.: Postoperative Massive Atelectasis: II. The Effect of Hyperventilation with Carbon Dioxide, J. A. M. A. **90**:1759 (June 2) 1928.

33. Peshkin, M. M.: Asthma in Children: III. The Incidence and Significance of Various Diseases and Infections, and of Tonsillectomy and Adenoidectomy, Am. J. Dis. Child. **33**:880 (June) 1927.

34. Peshkin, M. M.: Asthma in Children: I. Etiology, Am. J. Dis. Child. **31**: 763 (June) 1926.

35. Patients with para-asthma (a new term described by one of us [Dr. Peshkin] in the article referred to in footnote 34) cannot clinically be distinguished from those with other types of asthma; from the point of view of etiology, however, they must be differentiated from all other groups. These cases are caused directly by an enlarged thymus, by enlarged bronchial lymph nodes or both, and also by any other pathologic condition inducing tracheobronchial stenosis. The condition is sometimes referred to as obstructive asthma. This is an inadequate term, since the majority of subjects with these abnormalities do not show asthmatic symptoms. The patients acquiring para-asthma are inherently asthmogenic in type. The symptoms of asthma during the stage of para-asthma are continual as long as bronchial stenosis remains. After appropriate treatment, three patients with para-asthma, were relieved from all symptoms for periods ranging from two to four years. At the end of these periods symptoms recurred, and the condition was diagnosed as bronchial asthma of the protein nonsensitive type, because the factors responsible for the para-asthma were now absent.

asthmatic symptoms were not relieved in any of these patients by the injection of epinephrine hydrochloride (1 : 1,000 solution) as long as the partial stenosis was present.

The case reported in this paper is typical of para-asthma, the onset of asthmatic symptoms being initiated by some pathologic condition within the thorax—massive atelectasis. Early in the condition, the asthmatic râles were heard throughout the unaffected lung and that portion of the affected lung above the level of complete bronchial obstruction, while below the level of obstruction the breath sounds were entirely absent. Several days later, with only partial bronchial obstruction present, as shown by displacement of the heart and elevation of the diaphragm along with roentgenographic evidence of clearing of the pulmonary field of the affected side, asthmatic râles were heard for the first time throughout both lungs. Epinephrine hydrochloride and morphine sulphate failed to control the asthmatic signs in this stage of the disease as in the other instances cited of para-asthma due to other causes.

Massive atelectasis occurs more frequently in adults than in children. The cases occurring in children recorded in the literature are due to varied causes and include reports by Pasteur,[1] Elwyn and Girsdansky,[36] Abt,[37] Brennemann,[38] Griffith,[39] Wallgren,[5] Stoloff [40] and others.

The reported cases of massive atelectasis in patients with asthma are all in adults. The absence or rarity of this condition occurring as a complication of asthma in childhood is indeed noteworthy of our attention, particularly when one takes into consideration that in an experience of over ten years with a large series of children with asthma of whom roentgenograms were made as a routine measure at different intervals, not a single case of massive atelectasis was uncovered, in spite of the fact that these children have had various diseases and operations, especially that of tonsillectomy and adenoidectomy.

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36. Elwyn, H., and Girsdansky, J.: Postoperative Massive Collapse of the Lung: Report of Case, J. A. M. A. **79**:718 (Aug. 26) 1922.

37. Abt, I. A.: Massive Collapse of the Lung: Report of Case in a Boy Aged Ten Years, Am. J. Dis. Child. **30**:347 (Sept.) 1925.

38. Brennemann, J.: Etiology, Diagnosis and Treatment of Massive Collapse of the Lung and Allied Conditions, Wisconsin M. J. **26**:187 (April) 1927.

39. Griffith, J. P. C.: Massive Atelectasis (Massive Collapse) of the Lung, with Report of a Case Occurring in Infant of Three Weeks, M. J. & Rec. **123**: 103 (Jan. 20) 1926.

40. Stoloff, E. G.: Acute Massive Atelectasis of the Lung Complicating Fresh Tuberculous Primary Complex, Am. J. Dis. Child. **35**:239 (Feb.) 1928.

The case herein reported is of unusual interest in that it involves a condition of para-asthma in a child in whom the onset of the asthmatic state was initiated by acute massive atelectasis (nontuberculous and without surgical indication) of the left lung, and in whom, after the clearing of the atelectasis, the condition of true bronchial asthma was present for over one year. A careful search of the literature does not reveal a parallel case.

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PERSISTENT DUCTUS OMPHALOMESENTERICUS WITH SECONDARY ILEOSIGMOIDOSTOMY AND TERMINAL PERITONITIS *

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AND
FRANK R. MENNE, M.D.
PORTLAND, ORE.

Persistent ductus omphalomesentericus is of infrequent occurrence. In 1812, Meckel [1] described the involution of the ductus omphalomesentericus resulting in a persistent diverticulum. Since that time Cullen [2] has reviewed the literature concerning the irregularities of the involution of preumbilical structures. He reported forty-six cases of patent ductus omphalomesentericus. Most of these were observed at birth, though two cases were recognized when the patients were 16 and 28 years of age. Operations for the repair of the conditions were done in nineteen cases, resulting in fourteen recoveries and five deaths. Since that time, a number of similar or related anomalies of the umbilical cord have been recorded. Garratt [3] reported the persistence of vitelline ducts in twins. Other reports concerning persistent ductus omphalomesentericus have been made by O'Neil,[4] Barron,[5] Parker,[6] Guthrie,[7] Dietsen [8] and others. In all there have been published eighteen instances of the type of anomaly that is the subject of this report. Therefore, we regarded it of sufficient merit to add the details of another interesting case.

REPORT OF A CASE

History.—R. M., a white boy, aged 8, one of a family of six chlidren, all of whom, as well as the father and mother, were free from tuberculosis, had the following pertinent past history. He was a full term baby, normally delivered, weighing 9 pounds (4,082.33 Gm.) at birth. He was reared with difficulty, always

* Submitted for publication, Jan. 7, 1931.
* From the Departments of Pediatrics and Pathology of the University of Oregon Medical School, Portland.
* Read before the City and County Medical Society of Portland.

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2. Cullen, Thomas Stephen: The Umbilicus and Its Diseases, Philadelphia, W. B. Saunders Company, 1920.
3. Garratt, J. R.: Brit. M. J. **1**:645 (June 8) 1918.
4. O'Neil, W. E.: S. Clin. North America **3**:541 (June) 1919.
5. Barron, M.: Surg. Gynec. & Obst. **30**:350 (April) 1920.
6. Parker, C. H.: Am. J. Roentgenol. **10**:607 (Aug.) 1923.
7. Guthrie, D.: Boston M. & S. J. **190**:782 (May) 1924.
8. Dietsen: Ztschr. f. Chir. **201**:266, 1927.

having trouble with feeding, which was followed by acute pain. At 18 months he had had "intestinal influenza." This recurred when he was 2 years of age. Five months after this, he had an attack of "fever." At the age of 3, he was seen by a physician because of a large umbilical hernia and a large stomach. This physician observed many masses in the abdomen and made a diagnosis of tuberculous peritonitis. A tuberculin test at this time gave mildly positive results. During the next month the abdomen became progressively larger, and the umbilical hernia protruded more markedly. Finally the abdomen became hugely distended. During this time (nine days) of marked distention, there was no movement of the bowels. At that time the child was critically ill, but no history of vomiting or fever could be obtained from the parents. At the end of this time a large movement of the bowels was induced by repeated enemas. This was followed by rapid collapse of the abdomen. Following this, fecal material was observed to escape from the umbilicus for a period of one and one-half years. During this time, when the boy was from 4 to 4½ years of age, the bowels moved irregularly, and the umbilicus continued to drain; the draining gradually ceased. During this long period, the bowels moved only with the aid of enemas. The stool was usually very soft, occasionally it was normal. Following the closure of the umbilicus, the child improved and was able to run about and play with his friends. He started to attend school at the age of 6 years and attended regularly until his present illness. The child was seen at his home by one of us (Dr. Bridgeman) when he was 8 years of age. He had been sick for a week with what his mother thought was either a cold or a return of the former condition. At this time, the boy was found to be critically ill. He was breathing rapidly, was cyanotic and had an anxious expression.

Examination.—Physical examination revealed pneumonia in the lower lobe of the right lung and a greatly extended abdomen. The umbilicus was slightly protruding and mildly inflamed. A diffuse marked tenderness of the abdomen was observed, but noteworthy rigidity was not present. The child was admitted to the Doernbecher Hospital on April 26, 1930. At the time of admittance, his temperature was 102 F. It remained at this level for three days, when it rose sharply to 107 F. at the time of death. The pulse varied directly with the temperature, ranging from 150 to 190 F. The respiratory rate ranged between 55 and 70. The white blood count was 6,000, with 56 per cent polymorphonuclear leukocytes. The urine contained acetone, diacetic acid and albumin.

Course.—The patient was placed in an oxygen tent and treated symptomatically. Surgical consultation was held, but operation was deferred because of the serious condition of the patient. The patient died on April 29, three days after admission.

The clinical diagnosis was: tuberculous peritonitis with fistulous tract involving Meckel's diverticulum and terminal bronchial pneumonia.

Autopsy.—At autopsy, the following salient gross and microscopic changes were observed: The body was that of a white boy 8 years of age, 131 cm. in length and in a fair state of nutrition. A foul-smelling mucous material escaped from the nostrils, and the mouth contained a similar foamy, brownish material. There were puncture wounds in both upper extremities from the hypodermic needle. The body was symmetrical and well proportioned. The costal borders were flaring, and the abdomen was distended and tense. When the abdomen was opened, the great omentum was short and thick and extended for only a few centimeters below the transverse colon. The bowel was exposed. There was an accumulation of purulent material in the peritoneal cavity, which was most abundant from the umbilicus to the pelvic floor. The purulent fluid had a foul, putre-

factive odor. The small bowel was markedly extended; its walls appeared thickened and fibrous. Its caliber was equal to that of the large bowel. The lower end of the ileum was firmly fixed to the anterior surface of the sigmoid (fig. 1). This union of the ileum was 36 cm. from the ileocecal junction. Three cordlike structures radiated from this fusion of the ileum with the sigmoid; one of them (*A*) was found to be the greatly stenosed lower end of the ileum, and another was found to pass directly to the umbilicus (*B*). The umbilicus was surrounded by a dense mass of fibrous connective tissue, which was coated over with fibrin. The third branch extended upward, dividing into right and left strands (*C* and *D*). The cord (*C*) extended upward, branching (*E* and *F*) to the right again. The right branch (*E*) of this forking terminated in a hard, calcified nodule that was situated in the abdominal wall just below the costal border and in the midaxillary

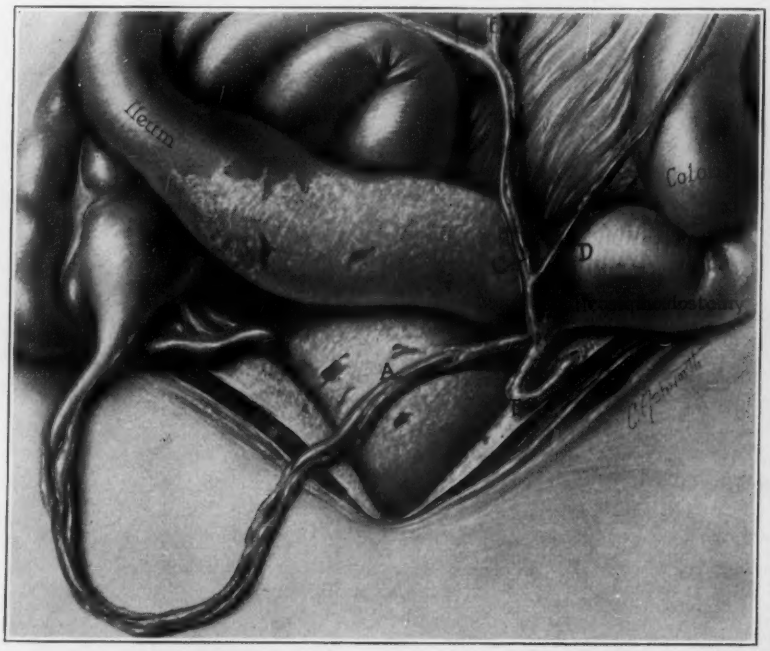

Fig. 1.—Drawing illustrating the appearance of the opened peritoneal cavity and the ileosigmoidostomy. *A*, indicates the distal portion of the ileum; *B*, the persistent ductus omphalomesentericus; *C*, *D*, *E* and *F*, radiating cordlike structures.

line on the right side. The other branch (*F*) extended to the right and terminated in the great omentum slightly to the right of the midline. At the primary division, the branch *D* extended upward to the left, terminating in a nodule that was similar in structure and location to that serving as the terminal for branch *E*. The portion of the ileum (36 cm.) beyond the ileosigmoidostomy was of small caliber, and its lumen immediately distal to this false union was only 0.5 cm. in diameter. The fistulous opening between the ileum and the sigmoid was also only 0.5 cm. in diameter (fig. 2). This opening was spanned by a bridge of mucosa. There was some ulceration with two small openings into the peritoneal cavity

at this point. This was evidently the site of the spread of the infection which resulted in the peritonitis. A few small polypi occurred in the mucosa of the sigmoid in this immediate region. The remaining portions of the wall of the bowel were intact. Both the ileum and the sigmoid had walls 0.8 cm. thick, which were uniformly hypertrophied. The serosa had an opaque, pearly gray appearance. Scattered adhesions of fibrous connective tissue were seen about the liver, spleen and sigmoid. No other anomalies or disease processes were observed in the examination of the rest of the body, except those mentioned in the anatomic diagnosis.

Microscopic examination of the ileum proximal to the ileosigmoidostomy revealed hypertrophy, edema and fibrosis throughout the wall, except the serosa which infiltrated with the products of acute exudation. These alterations were also found in the colon. Sections of the distal portion of the ileum disclosed atrophy of the entire wall with diffuse fibrosis. The serosa was also infil-

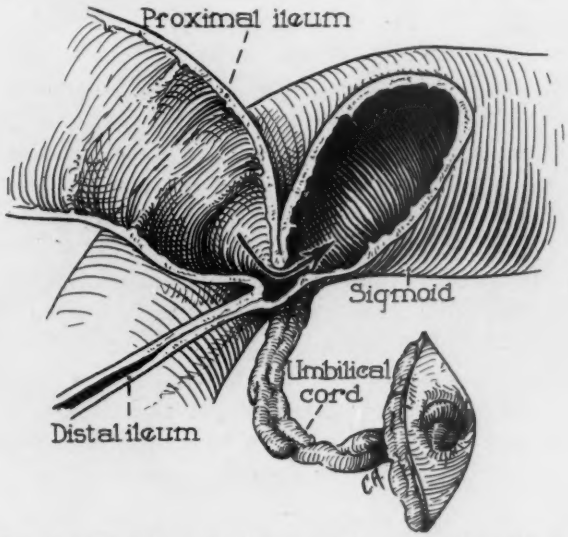

Fig. 2.—Drawing illustrates the character of the ileosigmoidostomy and the variation in the proximal and distal portions of the ileum.

trated with exudate. Microscopic sections of the remnant of the ductus omphalomesentericus and of the radiating cordlike strands disclosed similar structures. Partially or completely obliterated arterioles and veins occupied the center. The number of arterioles varied in different locations. There were usually two of these to one vein. The surrounding structure consisted of fibrous connective tissue and fat with many other small blood vessels and a considerable number of mononuclear phagocytes. The external or serous surface was coated with fibrin. The following anatomic diagnosis was made: acute serofibrinous peritonitis; spontaneous ileosigmoidostomy; local ulcerative iliosigmoiditis; persistent ductus omphalomesentericus; marked obliterative stenosis of the terminal portion of the ileum; chronic fibrous peritonitis; hypertrophy and dilatation of the proximal portion of the ileum and colon; acute interstitial bronchopneumonia of the lower lobe of the right lung; cloudy swelling of parenchymatous organs; hyperplastic pylorus, and patent foramen ovale.

COMMENT

The outstanding characteristics of this case of persistent ductus omphalomesentericus were those concerning the length of its duration, the mechanism of its production, the formation of the spontaneous ileosigmoidostomy and the fatal termination.

An analysis of the history bears evidence of the existence of a marked gastro-intestinal disturbance that manifested itself shortly after the infant's birth. It seems to us that the distress he exhibited after feeding was due not alone to the anomaly of the umbilicus, but in part to the hypertrophy of the pyloric end of the stomach. Later on in the course of the child's development, the persistent ductus omphalomesentericus began to cause certain disturbances that were probably due to infection. The condition was undoubtedly augmented by the persistence of a distinctive Meckel's diverticulum. It is our opinion that when the patient was 3 years of age, a definite Meckel's diverticulitis developed, which resulted in its fusion to the anterior surface of the sigmoid. This was accompanied by clinical evidence of obstruction and local peritonitis that lasted for nine days. The evidence of the existence of this condition was to be found in the location of the union (36 cm. from the ileocecal juntion), the absence of the omentum and its representation by the fibrous cords radiating from the ileosigmoidostomy and the scattered adhesions of fibrous connective tissue in various parts of the peritoneal cavity. At first it was thought that these fibrous cords might represent anomalous remnants of the vitelline ducts or umbilical veins. Subsequent microscopic study, however, revealed them to be remnants of the omentum which became adherent to the site of inflammation and subsequently absorbed except for the dense vascular fibrous cords seen at autopsy.

At the time of autopsy, except for the acute ulceration, the ileosigmoidostomy bore evidence of having existed for a long time. This is further substantiated by the marked hypertrophy and dilatation of the proximal portion of the ileum and the entire large bowel. That this union was the result of an acute Meckel's diverticulitis cannot be doubted, as such conditions are known to occur with all of the symptoms of obstruction or those of acute peritonitis. Hunter [9] and others have reported such occurrences in children.

It is of interest to note that following the spontaneous union of the ileum and sigmoid, which occurred about five years prior to the terminal illness, a greatly altered mechanical and physiologic activity of the bowel must have obtained. This is evidenced by the history of irregularity of the stool. The soft, mushy or watery character of the latter was

9. Hunter, Warren C.: Perforated Gangrenous Meckel's Diverticulum in a New-Born Infant, Am. J. Dis. Child. **35**:438 (March) 1928.

undoubtedly due to the modified or limited absorption of water by the colon. The irregularity of movement of the bowels was the result of altered rhythm in peristalsis and the dual passage of fecal material from the ileosigmoidostomy either by way of the terminal ileum before it contracted or through the new opening directly into the sigmoid. As the lower end of the ileum became smaller in caliber, reverse peristalsis must have been present, with varying effects on the absorption of water and the completion of digestion.

As the stenosis of the ileum progressed, the ileosigmoidostomy became exposed to greater trauma as the greatly hypertrophied proximal ileum attempted to force its contents through the opening. Partly because of this and partly because of efforts made to induce movement of the bowels, ulcerations with small fistulous openings developed, which lead to terminal peritonitis.

CONCLUSIONS

1. A case of persistent ductus omphalomesentericus in a boy, aged 8, is presented.

2. An acute Meckel's diverticulitis with stenosis of the lower end of the ileum followed by a spontaneous ileosigmoidostomy is delineated.

3. Such short-circuiting of the contents of the bowels leads to an unusual altered physiologic condition that may result fatally, due to peritonitis or obstruction.

ADHERENT CONGENITAL UMBILICAL HERNIA*

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True congenital umbilical hernia, described by other authors under the names exomphalos, omphalocele or hernia funiculi umbilicalis, is a relatively rare condition. It has been described as occurring about once in every 5,000 to 6,000 births, and it is apparently more common in the male than in the female.

As a type, it is differentiated from the true postnatal umbilical hernia and from the rare and more extensive defects of the abdominal wall that result in ectopy of the abdominal viscera. The latter class, which has also been described under the classification of fissurae abdominales, is not a true hernia, as there is no true umbilical ring but rather an extensive hiatus in the abdominal wall resulting in varying grades of malformation and eventration. The postnatal umbilical hernia so commonly seen differs from true congenital hernia in that it is more eccentrically situated about the umbilicus and in that it is characterized by a true ring formation and the usual coverings of an adult hernia. On the other hand, true congenital hernia exhibits a real ring formation and occurs directly through the center of the umbilicus; the covering, instead of being that usually found in a hernia, consists of the embryonal mesenchymal tissue, Wharton's jelly, that constitutes the umbilical cord.

It is hardly necessary to discuss at any length the theories concerning the causation of this condition. Several different explanations have been advanced by excellent observers. Each of these merits attention, and each probably represents one aspect of the condition. Aschoff [1] expressed the opinion that, along with other defects in the abdominal wall, this condition was explained best on the basis of a persistence of the fetal concavity of the dorsal spine. Kermanauer [2] attributed the defect to an arrest in the development of the primitive vertebrae and the abdominal walls. Ahlfeld [3] believed that the persistence of the omphalomesenteric duct was responsible, while others considered the presence of a fetal peritonitis as the underlying mechanism. Though I have not had sufficient experience with these conditions

* Submitted for publication, Jan. 17, 1931.

1. Aschoff, L.: Virchows Arch. f. path. Anat. **144**:511, 1896.

2. Kermanauer, quoted by Hannes, W.: München. med. Wchnschr. **58**:2664, 1911.

3. Ahlfeld, F.: Archiv. f. Gynäk. **11**:85, 1877.

to warrant critical discussion of the theories, it is interesting to note that in my case the liver was not found in the sac, and that a fairly large Meckel's diverticulum, as well as firm adhesions between the cecum and the umbilical ring, was found. Indeed, the adhesions prevented the preoperative reduction of the hernial contents.

REPORT OF CASE

H., a boy, seen in consultation with Dr. Gartner, on Sept. 5, 1929, five hours after birth, was a full term baby and had been born after a normal labor. On attempting to tie the umbilical cord, the physician had observed a large mass distending the cord for a distance of about 8 inches (20.3 cm.) from the navel. Realizing that he was faced with a true congenital hernia, the physician tied the cord at a distance of about 12 inches (30.4 cm.) from the abdominal wall and sought assistance.

When first examined the child was a normal, lusty baby, apparently not in distress, lying quietly in its crib. A sausage-shaped piece of umbilical cord about 12 inches in length projected from the umbilical region. Through the somewhat dimmed translucency of Wharton's jelly, several loops of small intestine could be seen in active peristalsis. With a sterile glove and under sterile precautions, an attempt was made gradually to reduce the intestinal mass within the abdomen by taxis. After several attempts in which practically all but one knuckle of intestine could be reduced, it was felt that operation was justified. Without anesthesia, a longitudinal incision was made into the umbilical cord and the peritoneal cavity was opened. Practically all of the intestine was reduced, when a fit of crying caused a recurrent prolapse of the intestinal contents. Ether anesthesia was then administered, and the incision was extended about 1 inch (2.5 cm.) upward on the abdominal wall. The hernial sac and its contents were now clearly exposed. The liver and other viscera, apart from the intestine, were not found in the hernial sac. The tip of the cecum was adherent to about two thirds of the circumference of the umbilical ring. The terminal loop of the ilium, to which a Meckel's diverticulum about 2 inches (5 cm.) in length was attached, was found lying in the sac.

To avoid injury to the intestine, the portion of the umbilical ring that was adherent to the cecal wall was cut away from the abdominal wall, leaving it attached to the intestine. Immediately the whole of the intestinal contents could be reduced without difficulty. The umbilical ring was closed with a purse-string suture of chromic catgut. The incision in the abdominal wall was closed with a through-and-through catgut stitch. The skin was closed with two interrupted silk stitches, leaving the umbilical cord in situ at its attachment to the abdominal wall. The cord was tied in the usual manner about 2 inches from the abdominal wall and the excess portion amputated. A dry dressing was applied. Apart from a fever which rose as high as 103 F. on the day following operation, probably due to inanition, the infant made an uneventful recovery and has since progressed in a normal manner without untoward results.

The interest in the consideration of such cases lies not only in their rarity, but in the prognostic significance of early surgical intervention as compared with conservative treatment. While it is true that occasionally gradual recovery may take place even when the patient is not treated, the usual outcome is necrosis of the cord, exposure

of the intestines and the development of peritonitis. In 1903, Lotheisen [4] reported the following results in ninety-one cases: conservative treatment: recovery, 34.78 per cent; mortality, 65.22 per cent; operative treatment: recovery, 70.6 per cent; mortality, 29.4 per cent. Finsterer [5] reported an operative mortality of 23.2 per cent and observed that in the operative cases the mortality increases with the lapse of time, as shown in the table. This remarkable increase in the mortality has

Mortality in Cases of Adherent Congenital Umbilical Hernia

Age of Patient	Number of Cases	Mortality
Up to 6 hours	14	0
Up to 12 hours	7	1
Up to 24 hours	12	5
Up to 36 hours	3	1
Up to 48 hours	4	2
Up to 5 days	1	0
Up to 6 days	1	0
Undetermined	3	1

been explained by some writers on the basis of the observations made by Tissier [6] that during the first ten or twelve hours after birth the intestine is completely free from bacteria, thus minimizing the chances of postoperative peritonitis.

2178 Broadway.

4. Lotheisen, G.: Wien. klin. Rundschau **17**:757, 1903.

5. Finsterer, J.: Wien. klin. Wchnschr. **19**:795, 1906.

6. Tissier, H.: Flore intestinale des nourrissons, Paris, Carré et Naud, 1900, p. 144.

Progress in Pediatrics

INTRAVENOUS UROGRAPHY IN INFANTS AND IN CHILDREN *

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AND
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Cystoscopy and pyelography in children may be difficult and usually requires the use of some form of a general anesthetic with its additional incidental dangers. The advent of intravenous urography was hailed with considerable enthusiasm, as it seemed that by this method diagnosis of disease of the urinary tract would be accomplished much more easily and safely than by the methods then in use.

It was hoped that it would be of especial value in children and infants by eliminating the difficulties and dangers attending the older methods of cystoscopy and retrograde pyelography. In particular it was hoped that a visualization of the urinary tract would be possible in infants and children who could not be subjected to cystoscopy with pyelography because of their size or on account of anomalies of the genito-urinary tract.

The following case reports with illustrations are presented to show the advantages, disadvantages and limitations of this new method as we have found them. All of the patients are less than 12 years of age. The cases are chosen from our series to show various groups of disorders of the urinary tract that occur in infancy and in childhood.

Our series covers the more common types of disease of the urinary tract seen in infancy and in childhood, as well as a number of types less commonly found. The accompanying table shows the pathologic conditions found in the 234 cystoscopic examinations immediately preceding our work with intravenous injections of urographic mediums. By comparison with this table, it will appear that the cases here reported represent a fair cross-section of the pathologic conditions of the urinary tract as encountered in infants and in children. We have not included all of our cases, nor have we taken as illustrative cases only those that gave the best visualization of the urinary tract following the intravenous injection of urographic mediums. The inferences that we have drawn from this series would not be appreciably changed by including all of

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the cases in all the different groups of diseases of the urinary tract. We believe that these cases represent what may be accepted at present, in our clinic, as a basis for our conclusions.

TECHNIC AND DOSAGE

In the spring of 1930, through the kindness of Dr. William C. Quinby of the department of urology of the Harvard Medical School, we were able to obtain a considerable amount of iopax before it had been actually placed on the market. With this supply we were able to determine to some degree the most suitable dosage of the drug, and the optimum time for obtaining films after its intravenous injection. This was of considerable economic value, as now the cost of the drug adds considerably to the financial burden put on the patient, as does the cost of taking a large number of films. In our earlier cases we took films immediately, and at least every fifteen minutes after the injection of the drug, up to two hours, and in some cases at even later intervals. In many cases the drug throws a shadow within a very few minutes after injection, but in studying our results for the purpose of economy in minimizing the number of films necessary, it would appear that the films that show the best concentration of the drug in the renal pelves and ureters are those taken about from forty-five to sixty minutes after injection. The drug is given in the x-ray room. The patient is put on the x-ray table in the Trendelenburg position with the bladder bag inflated, and exposures of from one twentieth to one tenth of a second are used. In these small patients we are unable to get enough cooperation by their holding their breath to obtain a satisfactory film by the longer and more desirable exposures. Therefore, it is not possible to use the Potter-Bucky diaphragm. Fluids are limited before and during the procedure. Catheterization of the bladder, before the films are taken from forty-five to sixty minutes after injection of the drug, may often result in better visualization of the lower end of the ureters.

The dosage as outlined by Swick was followed in most of our work, and the injection was given by the gravity method. As the small dose often threw no shadow when injected into some of the young infants, we tried larger doses in later cases. A full adult dose was given to patients under 1 year of age on three occasions, with no reaction. In only two cases was a reaction noted after the use of a proportionately large dose of the drug. For example, the patient in case 1, who was one of the smallest patients, weighing 12 pounds (5,443.1 Gm.), received 10 Gm. (one-fourth the adult dose) on one occasion, with no visualization of the kidneys or ureters. Some weeks later, after we had given considerably larger doses to other infants, we gave this patient a full adult dose without any demonstrable reaction, but also without obtaining satisfactory visualization of the kidneys or ureters. We had one death,

that of the patient in case 5, aged 6½ months, who was given three fourths of an adult dose. The patient presumably had considerable damage of the kidney, but it was not thought to be greater than in some of the other cases of this group. It is probable that almost any operative or manipulative procedure might have turned the scale and resulted in a fatal outcome. In this case, nonprotein nitrogen was 27 mg. per hundred cubic centimeters and the phenolsulphonphthalein test gave a reaction of 30 per cent in two hours. Although the laboratory observations were such as to indicate that there was not as severe damage of the kidney as in some other cases in which a proportionately larger dose of the drug was given, there was a fatal outcome within eighteen hours. While the cause of death cannot be definitely attributed to iopax, the premortal signs developed so shortly after its administration that this possibility cannot be disregarded.

In spite of the reports in the literature at that time regarding the nontoxicity of the drug, greater doses were given to these smaller patients only after careful consideration, and in the hope that better visualization of the urinary tract might result. At that time there was little information regarding the use of the drug for such small infants. In our experience severe reaction is unusual, but does not seem to be entirely dependent on the dosage of the drug or on the degree of damage of the kidney present, as shown by the clinical picture and the laboratory observations. On admission, the patient in case 4, aged 2 years, had a nonprotein nitrogen of 280 mg. per hundred cubic centimeters. Following drainage of the bladder, the nonprotein nitrogen dropped to 55 mg. At this time a reaction did not follow the administration of a full adult dose. There was also little, if any, visualization of the kidneys and ureters. A severe reaction is possible, though it is not usual or predictable to any certainty. As it seems established that no better visualization of the urinary tract is likely to be obtained by larger doses of the drug, it seems unwise in general to use them for these small patients. We now use approximately the proportionate doses as outlined in the original articles. In one of our more recent cases, however, a second larger dose was given as there was no visualization of the kidney and ureter when the smaller dose was used. The second and larger dose gave good visualization of the kidney. Skiodan was used in this case. In spite of poor renal function and the proved fact that there was but one kidney, no reaction followed the use of this drug on either occasion (case 21).

Except for the one fatal case (case 5), the only patient having an appreciable reaction was a boy (case 7), aged 6 years and 9 months, with obstruction at the outlet of the bladder, who after a full dose of iopax had a rise in temperature, flushing and some nausea and vomiting. These symptoms lasted for only a few hours. At the time of injection the nonprotein nitrogen was 25 mg. per hundred cubic centimeters.

REPORT OF CASES

CASE 1.—L. F., a boy, aged 9 months, was admitted to the hospital on June 1, 1930, with a condition diagnosed as megalo-ureter. He was poorly nourished, weighing 12 pounds (5,443.1 Gm.), and had a history of having been in another hospital for thirteen weeks with severe bronchopneumonia. Seven days before the present admission he became irritable, was feverish and refused food. This condition continued, and the child's temperature was about 103 F. Examination showed a poorly nourished, feverish child, who was restless and apparently in pain. His color was good. The lungs were clear. The heart and abdomen were normal. There was a moderate degree of rickets.

Repeated daily urinalyses showed a trace of albumin and a sediment loaded with pus cells. On July 1, the nonprotein nitrogen was 90 mg. per hundred cubic centimeters.

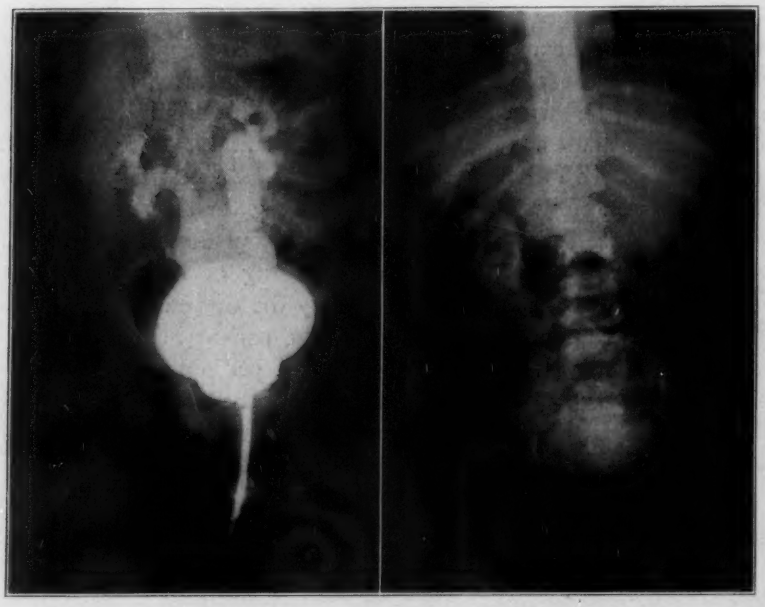

Fig. 1 (case 1).—The cystogram on the left was obtained after the injection of sodium iodide into the bladder. It shows well the enormously dilated and tortuous ureters, with some dilatation of the renal pelves. This should be compared with the cystogram on the right, which was taken after an intravenous injection of iopax. The opaque fluid is seen in the bladder, but only traces are visible in the kidneys and ureters. In both figures, one taken following one-fourth the adult dose and one following an adult dose, the visualization is unsatisfactory.

Pyelograms, taken after intravenous injection on June 9 (one-fourth the adult dose) and on July 1 (the adult dose), failed to show the renal pelves and ureters distinctly.

On July 7, suprapubic cystotomy was performed, with insertion of catheters into the ureters. What was thought to be a slight temporary improvement was followed by a gradual downhill course, with death on July 15.

The cause of death was pyelonephrosis. The other conditions included were: slight pneumonitis, acute splenitis, cloudy swelling of the liver, congestion of all the viscera and small hemorrhages into the lungs and kidneys. Complete microscopic study of the kidneys revealed nothing to indicate that the recently injected iopax had any effect on the existing severe damage of the kidneys. Examination and dissection of the genito-urinary tract after fixation in formaldehyde failed to show any anatomic constriction to explain the cause of the dilatation of the ureters and renal pelves. The verumontanum was not enlarged, and there were no valves or bands in the posterior urethra.

CASE 2.—T. M., aged 2 months, was admitted to the hospital on Feb. 18, 1930, with a condition diagnosed as megalo-ureter. The weight was 13 pounds and 14 ounces (6,293.6 Gm.). The patient was first admitted to the hospital on Dec. 22, 1929, because of fever, chills, refusal of food and convulsive twitchings of one week's duration. There was some evidence of nitrogen retention at this time, and the urine was loaded with pus cells. Cystograms taken after the injection of sodium iodide showed definite pyelo-ureteritis. On Feb. 20, 1930, a suprapubic cystotomy was performed, and at the same time catheters were placed in both ureteral orifices after dilatation. Subsequent to this and in association with pelvic lavage, the patient definitely improved. During one admission, pyelograms were attempted after the intravenous injection of a medium (June 12). The pyuria had persisted. The last admission was on December 8 for a slight nutritional disturbance. The patient was discharged on Jan. 1, 1931, weighing 24 pounds and 4 ounces (10,886.2 Gm.).

The urine contained from the slightest possible trace to a trace of albumin; the sediment, from 5 to 10 cells per high power field to a field loaded with white blood cells. The nonprotein nitrogen was 46 mg. per hundred cubic centimeters; phenolsulphonphthalein increased from 15 to 35 per cent in two hours and ten minutes.

Cystograms taken on Jan. 17, 1930, showed a well filled bladder, with reflux into both ureters, which were dilated and somewhat tortuous. The renal calices were somewhat blunted. On June 12, urography was performed after the intravenous administration of one-half adult dose of iopax. The renal pelves and ureters were not outlined, although the opaque material collected in the bladder in moderate concentration. This case was similar to case 1.

CASE 3.—M. N. was admitted to the hospital on July 30, 1930, because of vomiting, irritability, frequency and pyuria. The diagnosis was megalo-ureter. The weight was 10 pounds and 10 ounces (4,819.4 Gm.). Cystograms showed rather marked reflux up the ureters. The urine contained from a slight trace to a trace of albumin; the sediment was loaded with white blood cells. The nonprotein nitrogen was 33 mg. per hundred cubic centimeters; the phenolsulphonphthalein, 43 per cent in two hours and ten minutes.

Cystograms showed reflux in both ureters and renal pelves, with marked dilatation on both sides. On September 19, pyelograms taken by the intravenous method failed to show the renal pelves and ureters distinctly, although there was a moderate amount of the opaque material in the bladder (fig. 2).

CASE 4.—B. B., a boy, aged 2½ years, was admitted to the hospital on July 12, 1930, because of urinary frequency and dribbling most of his life. The diagnosis was urethral obstruction, pyelonephrosis and right inguinal hernia. The weight was 35 pounds (15,876 Gm.). The abdomen was constantly enlarged and distended. Before admission, the urine had become bloody, and micturition was painful. Examination showed a poorly developed and nourished child with essentially

negative findings, except for a markedly distended bladder, a palpable mass in the region of the right kidney, a hernia sac bulging into the right scrotum and almost continuous dribbling of blood-stained urine.

The urine was pink and contained a trace of albumin; the specific gravity was 1.006; the sediment showed gross blood and microscopic pus (admission). On admission, the nonprotein nitrogen was 280 mg.; on July 24, 120 mg., and on August 10, 54 mg. per hundred cubic centimeters. The white blood cells varied between 16,000 and 18,000.

CASE 5.—R. Q., a boy, aged 6½ months, was admitted to the hospital on Sept. 16, 1930, with a condition diagnosed as ureteral calculi and pyelonephritis. He

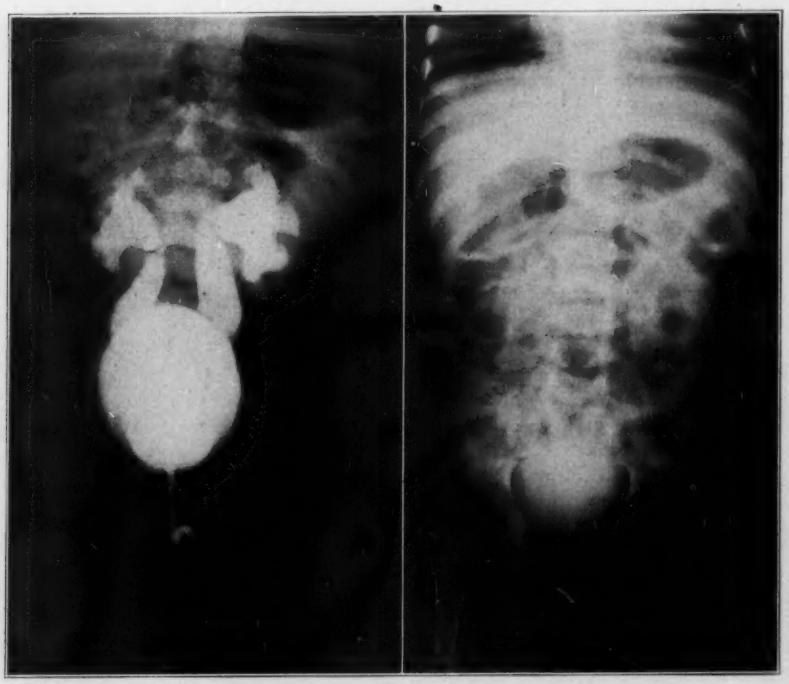

Fig. 2 (case 3).—The cystogram on the left was taken after the injection of sodium iodide into the bladder; it shows marked dilatation of both kidneys, pelves and ureters. The cystogram on the right was taken sixty minutes after the intravenous injection of iopax (three-fifths the adult dose); it shows only the collection of dye in the bladder.

was admitted for investigation of the genito-urinary tract. He weighed 12 pounds and 14 ounces (5,840 Gm.). The temperature had been 104 F. on several occasions during the two previous months, and there had been a loss in weight from 14 pounds and 6 ounces (6,520.3 Gm.) to that at the time of admission.

After admission there was a slight fall in temperature. About 30 Gm., or three-fourths the adult dose, of iopax was given. Following the injection, the patient became pale and listless. Parenteral fluids were given. In about six hours there was a gradual rise in temperature to 105 F. The pulse and respirations became more rapid. The condition became more severe, and the patient

died eighteen hours after the administration of the iopax. We were unable to obtain permission for a postmortem examination. We do know from the chronic course of pyuria, associated with high fever and a steady loss of weight over three months, that the patient had damage of the kidney, though the laboratory observations did not indicate as severe renal damage as in other cases in which larger doses had been used. However, the premortal signs developed so soon after the injection of a relatively large dose of iopax that its influence as the cause of death scarcely can be denied.

The phenolsulphonphthalein was 35 per cent in two hours and ten minutes. The urine was loaded with pus cells daily. The nonprotein nitrogen was 27 mg. per hundred cubic centimeters. A culture of the urine produced *B. coli.*

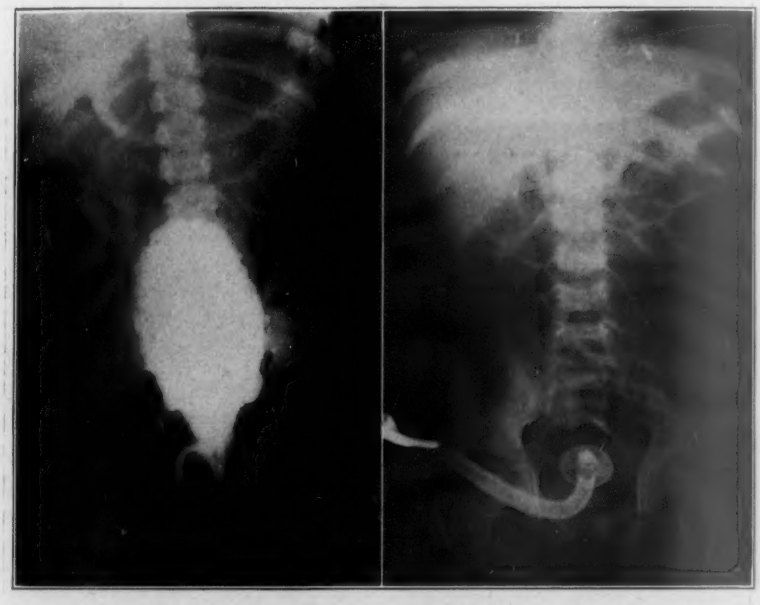

Fig. 3 (case 4).—The cystogram on the left shows the bladder outlined with sodium iodide. It has irregular, serrated margins, and there is dilatation of the posterior portion of the urethra. There is also what appear to be two small diverticulae on the inferior surface of the bladder. The figure on the right shows an attempted pyelogram after suprapubic cystotomy, by the intravenous method. There is no evident opaque dye in the kidneys, ureters or bladder. There was a suggestion of a trace after twenty-four hours. At the time the iopax (adult dose) was injected, the nonprotein nitrogen was 50 mg.; when the cystogram on the left was taken, it was 280 mg. per hundred cubic centimeters.

Case 6.—J. M., a boy, aged 6 years and 9 months, was admitted to the hospital on March 24, 1930, for urologic study. The diagnosis was congenital obstruction of the outlet of the bladder and hydronephrosis. He weighed 44 pounds (19,958.4 Gm.). A cystogram taken after the injection of sodium iodide showed a characteristic picture of obstruction at the outlet of the bladder (fig. 5). A suprapubic cystotomy was performed on April 11, and catheters were placed in the left ureter and bladder. The right pelvis was drained through an incision

in the right loin. Both ureters were filled with pus. The patient had a stormy recovery, but there was a fall in the nonprotein nitrogen of the blood and a steady increase in the output of phenolsulphonphthalein. On May 25, a portion of the obstructing tissue at the outlet of the bladder was removed with a Young punch. Following this procedure, the patient improved and was discharged from the hospital.

The patient was again admitted to the hospital six months later for further observation. Pyelograms were taken by the intravenous method. There was a definite reaction from the iopax. The patient's face became quite markedly flushed, and he vomited four or five times. The temperature rose to 101.8 F.;

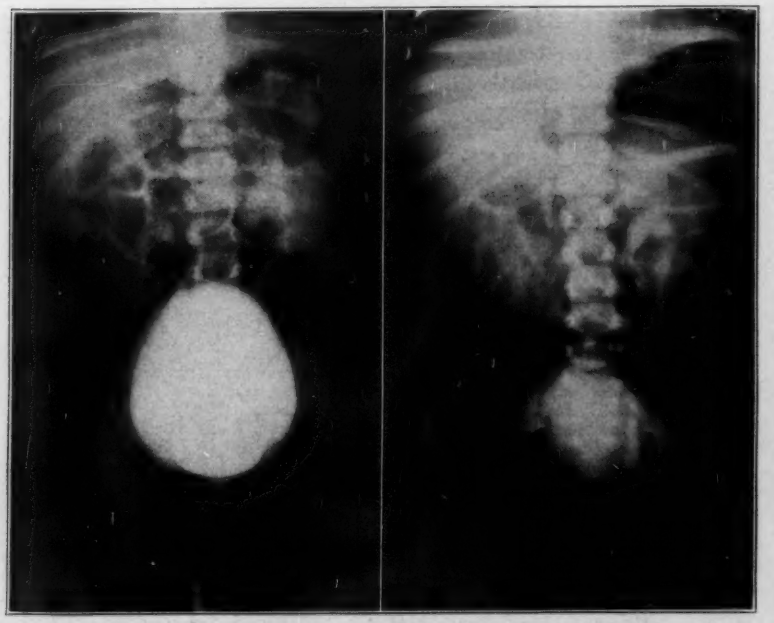

Fig. 4 (case 5).—The cystogram on the left shows a well filled bladder, with reflux into both ureters. Note the enlargement of the renal pelvis and the blunting of the calices on the left. The picture on the right (iopax, three-fifths the adult dose) shows what appears to be a chain of calculi filling the lower end of the left ureter for about 3 cm., obviously causing obstruction.

the pulse rate to 130. This was rapidly followed by a fall within a few hours to a temperature of 97 F. and a pulse rate of 100.

The urine contained from the slightest possible trace to a light trace of albumin; the sediment was loaded with white blood cells. On March 25, the nonprotein nitrogen of the blood was 80 mg. and on September 16, 41 mg. per hundred cubic centimeters. On March 27, the phenolsulphonphthalein was 15 per cent in two hours and on September 16, 40 per cent.

Case 7.—J. H., a boy, aged 6 years, was admitted to the hospital on Nov. 11, 1930, with exstrophy of the bladder. A right ureteral transplant had been carried out successfully in August. The left ureter was transplanted into the rec-

tum on November 14. Urograms were taken to demonstrate the condition of the kidneys and the pelvis following transplantation.

On November 19, the urine was normal. A blood count and a smear gave essentially negative results. The nonprotein nitrogen was 30 mg. per hundred cubic centimeters. Since his discharge from the hospital he had had an uneventful course at the convalescent home. The rectum emptied about every three hours.

In these cases, urography by the intravenous method offers a means of postoperative study heretofore not available by any method. Accordingly, a series of films was taken.

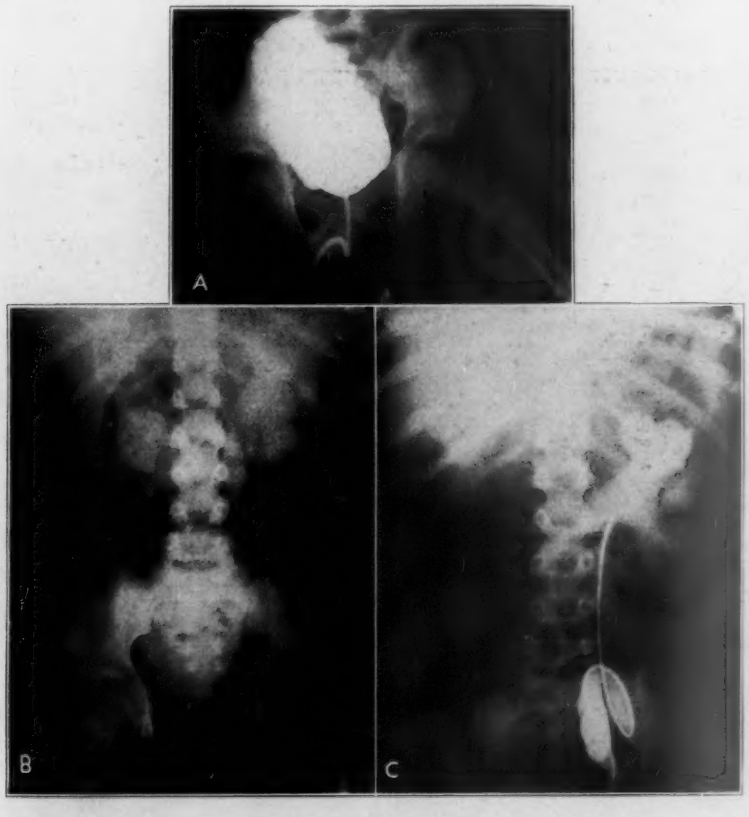

Fig. 5 (case 6).—*A*, the cystogram taken following the injection of sodium iodide into the bladder. The bladder is greatly distended and sacculated, and the appearance is the result of the obstruction at the outlet of the bladder. *B*, an intravenous urogram, showing the outline of the enormously dilated left renal pelvis, and the blunted calices. The dilated left ureter is well shown. There appears to be little opaque material on the right. Compare this with the pyelogram. (*C*) obtained by the retrograde catheterization, which outlines the renal pelvis much more distinctly. It also shows the much dilated and extremely tortuous left ureter more distinctly.

Sept. 6, 1930: After operation on the right side, a cystogram taken following the intravenous injection of iopax (one-half the adult dose) showed the renal

pelvis and calices rather faintly outlined, those on the left being normal and those on the right, sacculated and somewhat dilated.

Dec. 24, 1930: After operation on the left side, a cystogram taken following the intravenous injection of iopax (one-half the adult dose) showed moderately well outlined, dilated, renal pelvis, calices and ureter on the right. No dye was seen on the left.

Dec. 30, 1930: Another injection of iopax was given (two-thirds the adult dose). We were still unable to discern dye coming through on the left side.

Jan. 8, 1931: Five minutes after the intravenous injection of skiodan (full adult dose), the dye faintly outlined the right ureter and the renal pelvis. Subsequent films, taken after fifteen, thirty, forty-five and ninety minutes, showed the

Fig. 6 (case 7).—*A*, film (iopax) taken subsequent to transplantation of the right ureter. The left side is apparently normal; the right side shows sacculated and dilated pelves and calices. *B*, film (skiodan) showing the well outlined, dilated right ureter, pelvis and calices. The left kidney is well outlined, but with no dye coming through.

best concentration after thirty and forty-five minutes. The left kidney was well outlined and questionably enlarged, but dye was not coming through.

CASE 8.—F. S., a boy, aged 16 months, was admitted to the hospital on June 5, 1930, with the diagnosis of multiple congenital anomalies of the genito-urinary tract. The weight was 17 pounds and 8 ounces (7,937.8 Gm.). The anomalies consisted of two bladders, two penes, two scrota, hypospadias on the right and left, diastasis recti with a large ventral hernia and exstrophy of the lower part of the bladder. The urethra of the right penis led to the upper part of the bladder. No probe could be passed through the left penis, and no urine appeared from it (fig. 7).

Fig. 7 (case 8).—Photograph showing some of the congenital anomalies.

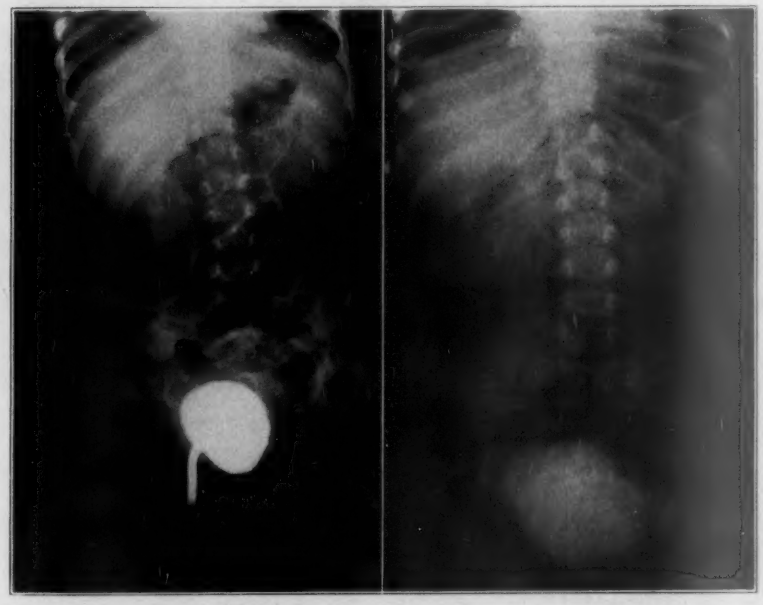

Fig. 8 (case 8).—The cystogram on the left was obtained after the injection of sodium iodide into the upper nonexstrophied bladder. There is a small amount of reflux to the right kidney. The figure on the right shows the opaque fluid (iopax) in the bladder, with a suggestion of the dye in the region of the right kidney. None is seen in the left kidney. There is no delineation of the ureters.

CASE 9.—S. H., a girl, aged 7½ years, was admitted to the hospital on July 2, 1930, for study. She weighed 63 pounds (28,576.8 Gm.). The diagnosis was double ureter on the left. Except for the double ureter, nothing abnormal was found, and this condition had no apparent relation to the symptoms.

The urine was normal, except for the slightest possible trace of albumin. Examination of the blood showed: red cells, 4,700,000; white cells, 10,200; polymorphonuclears, 64; lymphocytes, 34; monocytes, 4; eosinophils, 1, and basophils, 1. The phenolsulphonphthalein was 60 per cent in two hours and ten minutes.

CASE 10.—F. C., a boy, aged 10 years, was admitted to the hospital on April 24, 1930, with a condition diagnosed as tuberculosis of the kidney. The weight was 72 pounds (32,659.2 Gm.). He had had a tuberculous right kidney removed one and a half years before. During the interval following nephrectomy, he had

Fig. 9 (case 9).—The pyelogram on the left was obtained by the retrograde method. The urogram on the right was obtained after the intravenous injection of iopax. The one on the right seems to give a clearer picture of the anomaly, although visualization of the renal pelves is not good. In the intravenous urogram, the ureters on the left appear dilated. The patient exhibited no symptoms, and there was no pus in the urine.

done remarkably well and had gained 24 pounds (10,886.4 Gm.). Six weeks before the present entry, he began to have painful and frequent urination. On admission, he was passing bloody urine in small amounts frequently. Cystoscopy was not possible on account of the irritable urethra. Pyelograms were made after intravenous injections.

CASE 11.—D. P., a girl, aged 7½ years, was admitted to the hospital on Sept. 2, 1930, because of dull pain and tender swelling in the right upper quadrant of the abdomen. The diagnosis was primary carcinoma of the liver. The symptoms had been noted for five weeks. The patient weighed 52 pounds (23,587.2 Gm.). On examination, the important finding was a large mass in the right flank and

Fig. 10 (case 10).—Films taken after the intravenous injection of iopax showed the left renal pelvis dilated, blunted calices and a dilated left ureter. This film, taken with the history of tuberculosis, might justify a diagnosis of tuberculosis of the remaining left kidney. The patient's right kidney had been removed one and a half years before.

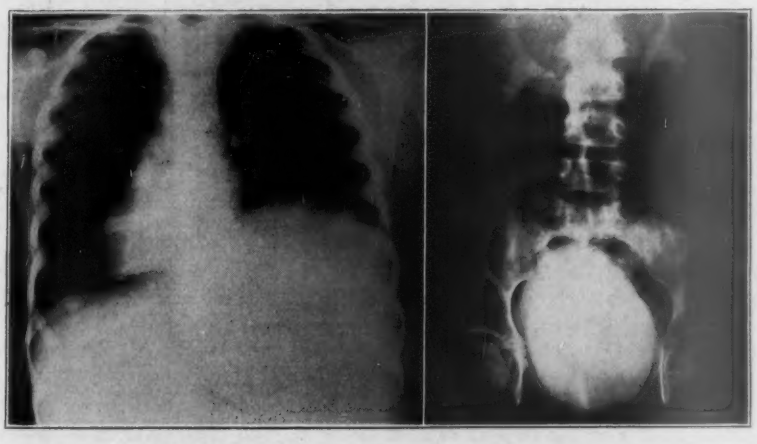

Fig. 11 (case 11).—The pyelograms, obtained by the intravenous iopax method, demonstrate normal kidneys. The film of the chest shows the great upward displacement of the diaphragm on the right. In this case iopax clearly established the differential diagnosis between new growth of the liver and new growth of the kidney.

upper quadrant which extended across to the left side and downward to the level of the umbilicus. This suggested a malignant condition of either the kidney or the liver.

Urinalyses gave negative results. Examination of the blood showed: hemoglobin, 70 per cent; red cells, 4,220,000; white cells, 16,750; smear, negative; Wassermann reaction, negative; tuberculin reaction, negative; nonprotein nitrogen, 36 mg. per hundred cubic centimeters; total protein, 5.9; culture, negative; icteric index, 7; bleeding and clotting time, normal.

On September 8, exploratory laparotomy revealed a large intrinsic tumor of the dome of the liver in the right lobe. A specimen was taken for pathologic examination, which showed carcinoma of the liver of hepatic cell origin.

Fig. 12 (case 12).—The intravenous administration of iopax gave this picture of congenital, bilateral, polycystic kidneys and elongated, trumpet-shaped and enlarged calices, evidently encroached on by the enlarged cysts of the kidneys.

CASE 12.—R. E., a white boy, aged 2 years and 11 months, was admitted to the hospital on Oct. 8, 1930, because of vomiting and failure to develop. The diagnosis was bilateral polycystic kidneys and diffuse cortical atrophy. He weighed 25 pounds (11,340 Gm.). Convulsions began when the child was 4 months old and persisted almost daily. Physical examination showed a poorly developed and nourished boy, who ruminated and sucked the thumb continuously. The head was large. The eyegrounds were normal. The abdomen showed large, palpable, lobulated masses in the regions of both kidneys. The reflexes were hyperactive.

The blood, urine and spinal fluid were normal. The Wassermann reactions of the blood and spinal fluid were negative. The reaction to a 1:1,000 solution of tuberculin was negative. The nonprotein nitrogen was 43 mg.; the serum protein, 45 Gm. per hundred cubic centimeters. The phenolsulphonphthalein was 35 per cent in two hours.

Roentgenograms of the extremities showed slender, atrophic long bones with multiple transverse lines near the ends of the diaphysis due evidently to repeated disturbances of growth. The skull, chest and spine were normal. Ventriculo-encephalograms showed markedly dilated ventricles, with an excess of air in the subarachnoid spaces.

Of interest in connection with this case is the fact that a sister, R. E., aged 4¼ years, who was admitted to the hospital, and discharged at the same time, presented the same symptoms. Ventriculo-encephalograms demonstrated cortical atrophy. Urograms (iopax, one-half the adult dose, being used) also showed almost the same picture as that described.

Case 13.—S. T., a boy, aged 11 years, was admitted to the hospital on July 29, 1930, because of slight pyuria and albuminuria of two months' duration. The

Fig. 13 (case 13).—The figure on the left, taken following an intravenous injection of iopax, shows both kidneys quite well outlined, but normal calices and ureters. The figure on the right was taken immediately after the one on the left, when the patient was in the upright position. Both kidneys and ureters show rapid and complete emptying.

diagnosis was orthostatic albuminuria. He weighed 69 pounds (31,298.4 Gm.). The physical findings were negative, except for an undescended left testicle. A test for orthostatic albuminuria was performed, with positive results. Urograms were made by the intravenous method.

The urine, after standing for ten minutes, showed a large trace of albumin; after rest in bed, it was negative for albumin. The nonprotein nitrogen was 33 mg. per hundred cubic centimeters; the serum protein, 5.34 Gm. The phenol-sulphonphthalein was 50 per cent in two hours.

Case 14.—M. M., a girl, aged 9 years and 8 months, was admitted to the hospital on Aug. 12, 1930, with a history of intermittent attacks of abdominal pain

during the past three years. The diagnosis was subacute appendicitis. She weighed 64 pounds (29,030.4 Gm). Roentgen examination and tests with tuberculin were negative. The results of physical examination were essentially negative, except for slight tenderness of the abdomen on deep palpation. The temperature was 99 F.

Repeated urinalyses gave negative results. The phenolsulphonphthalein was 50 per cent in two hours. Examination of the blood showed: hemoglobin, 90 per cent; red cells, 4,800,000; white cells, 13,000; polymorphonuclears, 69 per cent, and lymphocytes, 27.

Fig. 14 (case 14).—These pyelograms, obtained after the intravenous injection of iopax, show no urinary pathologic changes, although the films are not as distinct as some of those in other cases, and are not reliable enough to exclude by themselves any pathologic condition of the urinary tract.

Appendectomy was performed on August 16. The pathologic report was as follows: subacute appendicitis; the lymph follicles of the appendix showed infiltration with polymorphonuclear lymphocytes.

CASE 15.—R. McE., a boy, aged 10 years, was admitted to the hospital on Oct. 18, 1930, because of abdominal pain of five days' duration. The diagnosis was acute pyelitis (mild). There had been no nausea or vomiting. Frequency had recently been noted, but was not associated with dysuria. Physical examination

gave negative results, except for slight tenderness in the right lower quadrant of the abdomen. On admission the temperature was 103 F.; the pulse rate, 90, and the respiratory rate, 20. The patient weighed 70 pounds (31,752 Gm.).

The urine contained from 3 to 4 white blood cells per high power field, with occasional clumps of from 5 to 15 white blood cells. The white blood cells numbered 15,500. The reaction to a 1:1,000 solution of tuberculin was negative.

There was a gradual fall in temperature to normal, associated with disappearance of the symptoms. The patient was discharged from the hospital on October 25.

Fig. 15 (case 15).—This picture was taken following the injection of iopax; it demonstrates nothing abnormal.

CASE 16.—S. B., a girl, aged 10 years, was admitted to the hospital on Sept. 30, 1930, because of vomiting and abdominal pain of one week's duration. The diagnosis was pyelonephritis. She weighed 68 pounds (30,844.8 Gm.). She had had three or four attacks of dull pain in the past few months which incapacitated her for a few days. On examination, she presented little except slight costovertebral tenderness and pain in the right lower quadrant of the abdomen. Rectal examination gave negative results. The temperature on admission was 99.5 F. The diagnosis considered on admission was subacute appendicitis.

Examination of the blood showed: hemoglobin, 80 per cent; red cells, 5,900,000; white cells, 12,000; polymorphonuclears, 70; lymphocytes, 26; monocytes, 3. The

phenolsulphonphthalein was 70 per cent in two hours. The nonprotein nitrogen was 40 mg. per hundred cubic centimeters. The reaction to a 1:500 solution of tuberculin was negative. The urine showed a trace of albumin; the sediment was loaded with white cells.

The urine gradually cleared up following the administration of hexylresorcinol and forcing of fluids. The patient was discharged from the hospital relieved on October 6.

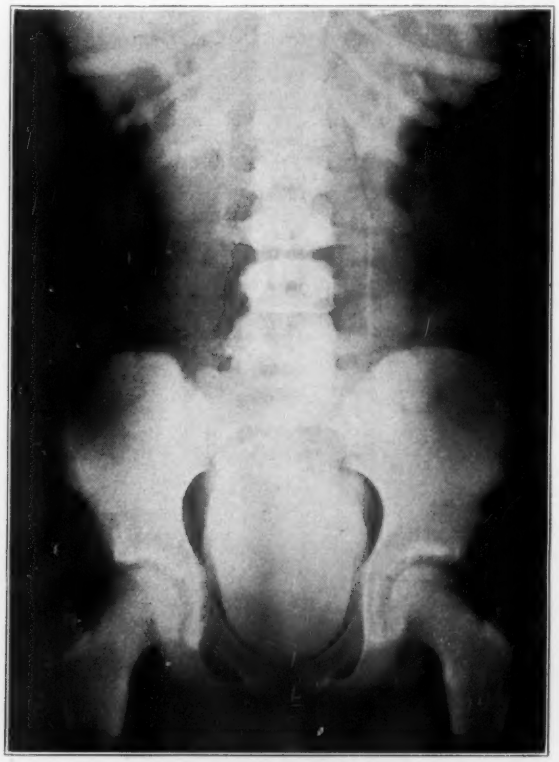

Fig. 16 (case 16).—This pyelogram was obtained by the intravenous method; it shows no abnormal observations.

CASE 17.—J. C., a boy, aged 10 years, was admitted to the hospital on July 29, 1930, because of several attacks of hematuria associated with pain in the left groin. The diagnosis was obstruction of the left ureteral orifice, pyelo-ureteritis and renal calculus. He weighed 62 pounds (28,123.2 Gm.).

The urine contained much pus; the nonprotein nitrogen was 49 mg. per hundred cubic centimeters; the phenolsulphonphthalein was 50 per cent in two hours.

CASE 18.—E. D., a girl, aged 9 years, was admitted to the hospital on Oct. 31, 1930, because of dysuria and frequency of two weeks' duration. The diagnosis was acute pyelonephritis. She weighed 58 pounds (26,308.8 Gm.). The patient was treated by urinary antisepsis and forced fluids for two weeks, with definite but not

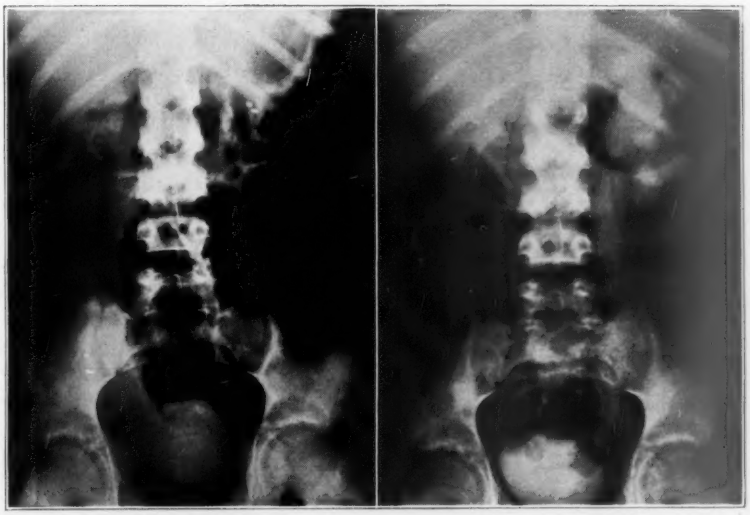

Fig. 17 (case 17).—The figure on the left (a flat film of the abdomen) shows two rounded, dense shadows overlying the area of the left kidney. The figure on the right is an intravenous urogram obtained after a suprapubic cystotomy with dilatation of the left ureteral orifice had been performed. There is enormous dilatation of the left kidney and ureter. The obstruction at the left ureteral orifice is the cause of the dilatation above, with stasis, infection and the formation of stones.

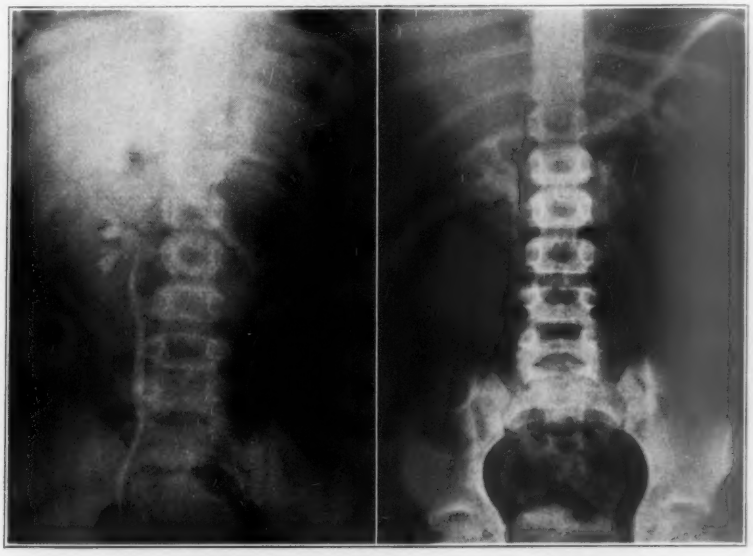

Fig. 18 (case 18).—The figure on the left is the pyelogram obtained with sodium iodide by ureteral catheterization. On repeated examination, the filling defect at the ureteropelvic junction persisted, as did a filling defect of the upper calix; an abnormality in this region could not be ruled out. The figure on the right was obtained following the intravenous injection of iopax. The film apparently rules out pathologic conditions in the right kidney and ureter.

complete relief. Cystoscopy for the purpose of diagnosis and pelvic lavage was advised. Because of the unusual cystoscopic observations, intravenous urography was also advised.

The urine contained from none to a trace of albumin; the sediment was loaded with white cells and clumps. The blood was normal. The reaction to a 1:500 solution of tuberculin was negative. The nonprotein nitrogen was 33 mg. per hundred centimeters. The phenolsulphonphthalein was 65 per cent in two hours. On November 6, a culture of urine from the bladder showed *B. coli.* On November 14, a specimen from the right and left kidneys was negative.

Retrograde pyelograms taken on November 17 showed a well filled, apparently normal renal pelvis and calices on the left. The ureter was not dilated. On the right, the upper calix did not appear to be filled, which suggested that there was

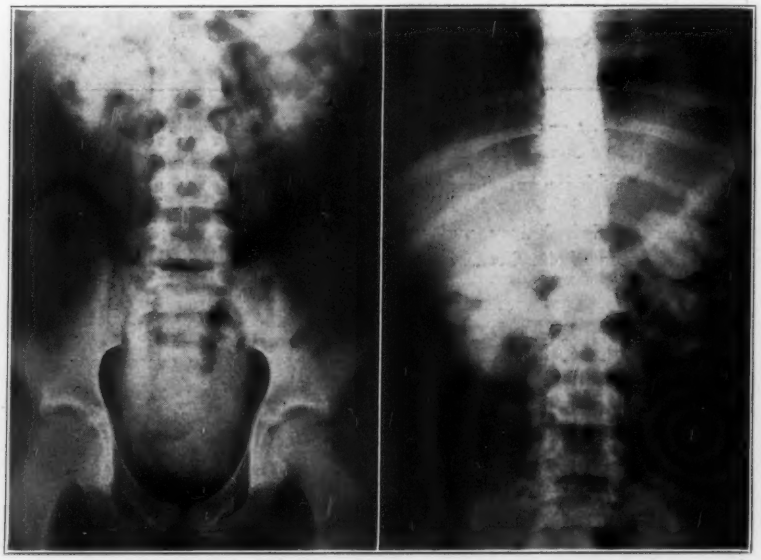

Fig. 19 (case 19).—Urograms (iopax, 40 Gm.), showing quite well filled but considerably dilated pelves with bluntness of the calices. The ureters are also markedly dilated. The visualization is good. Compare with figures 3 and 5.

probably another ureter supplying the upper pole. There was also a question of abnormality at the ureteropelvic junction on the right.

CASE 19.—M. B., a boy, aged 10 years, was admitted to the hospital on Sept. 24, 1930, because of dribbling and frequency since birth. There were also dysuria, headaches and visual disturbances of three weeks' duration. The diagnosis was congenital obstruction of the posterior urethra and pyelonephrosis. The symptoms became worse shortly after admission. Attempts at catheterization were unsuccessful, obstruction being met in the posterior urethra. A suprapubic cystotomy was performed, and congenital valves of the posterior urethra were demonstrated. These were excised by Young's punch two days later. (Following subsequent dilatations the patient became symptom-free and was discharged to the outpatient department on November 10. He returned in two weeks for dilatation and intravenous urography.)

On September 25, the urine was cloudy with a specific gravity of 1.010, and contained the slightest possible trace of albumin; the sediment showed many pus cells and motile bacilli; a culture showed *B. coli*. On September 27, examination of the blood showed: hemoglobin, 70 per cent; red cells, 4,650,000; white cells, 34,600; polymorphonuclears, 80 per cent; Wassermann reaction, negative. The nonprotein nitrogen was 95 mg. per hundred cubic centimeters; the serum protein, 9 Gm. On October 6, the nonprotein nitrogen was 39 mg. and the serum protein, 8 Gm. per hundred cubic centimeters.

CASE 20.—S. B., aged 18 months, was admitted to the hospital on Jan. 23, 1931. The weight was 16 pounds (7,263.4 Gm.). The diagnosis was hydronephrosis on the right and congenital absence of the left kidney. The patient had had marked pyuria associated with fever and failure to do well for six weeks. A cystogram

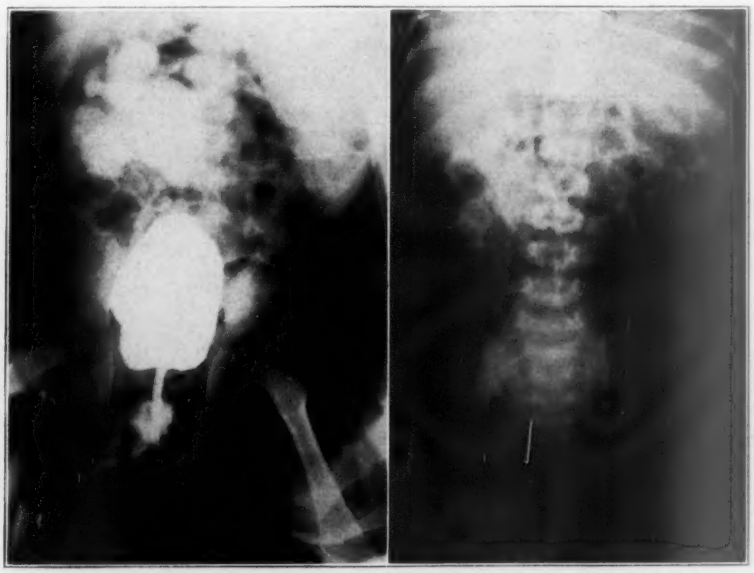

Fig. 20 (case 20).—This was the type of case from which one would hope to get the most information by the intravenous use of urographic mediums. A left ureter could not be found at cystoscopy, hence the absence of a left kidney was suspected. However, even two thirds of an adult dose of skiodan failed to differentiate clearly between the left and right sides. Exploratory laparotomy confirmed the cystoscopic diagnosis, i. e., congenital absence of the left kidney. The figure on the left is a cystogram; that on the right was taken following the intravenous injection of skiodan.

showed reflux up the right ureter and marked hydronephrosis. Removal of the right kidney was advised if the left kidney could be demonstrated. Skiodan (10 Gm., or one-half the adult dose) was given on Jan. 15, 1931, and (16 Gm., or three-fourths the adult dose) on January 19, without demonstrating signs of a left kidney.

The urine contained a trace of albumin and was loaded with white blood cells. The nonprotein nitrogen was 33 mg. per hundred cubic centimeters. The phenolsulphonphthalein was 10 per cent in two hours and 10 minutes. The blood urea clearance was 20 per cent.

On Jan. 10, 1931, a retrograde pyelogram showed a large renal pelvis and calix on the right side. The ureteral orifice on the left was not seen.

On January 15, urograms taken after the intravenous method (one-half the adult dose of skiodan) failed to show the renal pelvis or the ureters definitely on either side.

On January 19, the intravenous administration of skiodan (two-thirds the adult dose) before urograms were taken failed to outline the renal pelves and calices distinctly on either side.

On January 23, exploratory laparotomy confirmed the absence of the left kidney.

CASE 21.—D. G., aged 3 years and 10 months, was admitted to the hospital on Dec. 26, 1930. The diagnosis was complete epispadias, exstrophy of the bladder

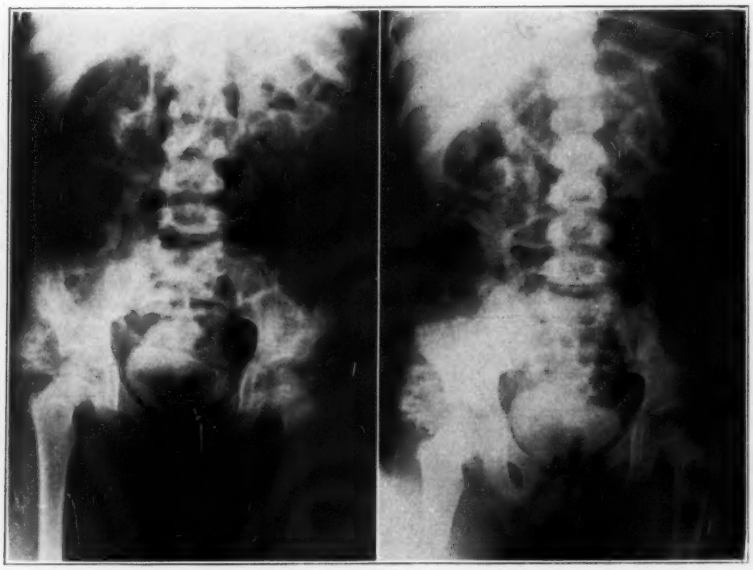

Fig. 21 (case 21).—Postoperative urograms (skiodan) taken on Jan. 13, 1931; they are indistinct due to gas, but show evidence of dye coming through on both sides with considerable accumulation of opaque material in the rectum as well as in the bladder. The film on the left shows the normal left pelvis; that on the right, an indistinct and slightly dilated right pelvis and ureter.

and transplantation of the ureters into the rectum. A transplantation of the right ureter into the rectum (Coffey technic) was performed on December 31. There was complete recovery, without a febrile reaction.

On December 27, the urine was normal. A blood count and smear were negative. The nonprotein nitrogen was 29 mg. per hundred cubic centimeters.

SUMMARY

The mediums used for urography by the intravenous method are valuable adjuncts in the diagnosis of disease of the urinary tract. With

improvement in the mediums and in the technic of administration, the value of this procedure will doubtless increase. It seems advisable, however, to point out certain definite limitations of its use in children, and especially to urge that the time has not yet come for its general use by the practitioner of pediatrics who is unfamiliar with urological procedures, and especially by one unfamiliar with the interpretation of the urograms obtained in young patients.

*Diagnoses Made in 234 Consecutive Cystoscopic Examinations**

Pyelonephrosis and pyelo-ureteritis	103		
Hydronephrosis			
Bilateral	5		
Right	7		
Left	10		
	22		
Tuberculosis of the kidney	3	Pathologic changes in the kidneys:	
Tumor of the kidney	2	Associated with infection of gross or moderate degree	130
Horseshoe kidney	2	Other	21
Polycystic kidney	3	Total	151
Ptosis of the kidney	3		
Renal calculus	9		
Nephritis	4		
Ureterovesicle cyst	1		
Ureterocele	1		
Double ureters			
Right	2	Ureteral pathologic changes	14
Left	7		
Bilateral	1		
	10		
Ureteral calculi	2		
Cystitis	16		
Exstrophied bladder	1	Pathologic changes in bladder	20
Vesicle calculi	2		
Bladder diverticulum	1		
Urethral obstruction	6	Urethral pathologic changes	6
Orthostatic albuminuria	2		
Phosphaturia	1	Symptoms of unknown pathology	12
Pyuria (cause not determined)	6		
Hematuria	3		
Chronic appendicitis	9	Gastro-enteric pathology	12
Mesenteric adenitis	3		
No diagnosis recorded			19
Total			234†

* Prepared by Mr. D. W. MacCollum, fourth year student, Harvard Medical School, as part of his thesis for his course in the surgery of children.

† It will be seen that in 160 of 234 cases cystoscopic examination showed infection of the urinary tract of marked or moderate degree. This is about 60 per cent of the entire series and does not include those with mild infection.

The foregoing cases are illustrative of the majority of the types of genito-urinary disease encountered in infancy and in childhood. It will be seen and has already been stated by others that cases associated with considerable infection of the urinary tract and concomitant renal damage may not give a satisfactory visualization of the urinary tract by the use of urographic mediums injected intravenously. However, in our experience and as shown by the accompanying table, the greatest number of cases in infants and children requiring urological investigation are those in which there is a considerable degree of pyuria and asso-

ciated renal damage. In such cases, for the most part, and notably in the pyurias of infancy, in which retrograde pyelography is impossible, intravenous urography has given either no visualization of the kidneys and ureters or so little that reliable information was not obtained. Cases of megalo-ureter of unknown cause and associated with pyuria form a fairly large group of the disorders of the urinary tract seen in early life. In these cases, cystography usually gives excellent visualization of the entire urinary tract; intravenous urography gives little or none (cases 1, 2 and 3).

In cases of congenital obstruction of the outlet of the bladder (cases 4, 6 and 19), with marked pyuria and associated renal damage, little, if any, visualization of the kidneys and ureters was obtained by intravenous urography (the patient in case 4, aged 2 years, and the one in case 6, aged 6 years); however, in case 19 (aged 10) good visualization was obtained.

There is a large group of cases, about 60 per cent, of diseases of the urinary tract in children that are associated with varying degrees of pyuria (see table 1). The present mediums for intravenous urography may give little, if any, reliable information if the pyuria and associated renal damage are marked. In addition, there may be a definite danger in its use. At present we are unable to predict from the information obtained by the clinical picture and by various tests of renal function which cases will show a reaction (cases 1, 2, 3, 4, 5 and 21).

In some cases of congenital anomaly of the urinary tract, intravenous urography alone will establish the diagnosis (case 9, with a double ureter, and case 12, with bilateral polycystic kidneys).

In other anomalies (case 8), double bladder, with exstrophy of one bladder and epispadias, the information was not conclusive as to whether there was one, two or more kidneys, nor was there any visualization of the ureters of either bladder. In cases 7 and 19, in which there was exstrophy of the bladder following transplantation of the ureter, intravenous urograms gave little definite information. However, this is the only method at present available for obtaining visualization of the kidneys and ureters in such cases.

In the group of cases showing only mild infection of the urinary tract, good visualization is generally obtained by this method. It does not yet give sufficiently good delineation of the urinary tract, however, for differentiation between the clinical picture of acute pyelitis that simulates an acute condition of the appendix and the case of subacute or acute appendicitis in which a mild pyuria may be present (cases 14, 15 and 16).

In our experience, intravenous urography is not a sufficiently reliable in cases of unilateral or bilateral pyelo-ureteritis of moderate severity. guide in determining the need for continued treatment by pelvic lavage

Of course, the information obtained by retrograde pyelography alone is not sufficient in these cases. The symptoms, the duration, the urinary observations, etc., must be considered.

In case 11, intravenous urography established the differential diagnosis between new growth of the kidney and new growth of the liver.

In case 10, cystoscopy and retrograde pyelography were impossible; tuberculosis of the remaining kidney was demonstrated by intravenous urography. The diagnosis could hardly have been missed in this particular case, from the history and physical examination alone. It would seem, however, that intravenous urography is of considerable value in such cases.

Cases of urinary calculus, especially those with obstruction of urinary outflow, generally give good visualization of the urinary tract, and in many cases retrograde pyelography may be dispensed with.

In case 16, intravenous urography gave definite information that had not been obtained by repeated retrograde pyelography, demonstrating no abnormality at the ureteropelvic junction or in the upper calix of the kidney.

In case 21, the usual intravenous dose of the dye (skiodan) gave no positive information as to whether there were one or two kidneys. It is our experience that in the presence of severe damage of the kidneys no shadow of the kidneys may be obtained after administration of the drug. It was of great importance to determine whether the hydronephrotic kidney on the right was the only kidney. On cystoscopic examination, no left ureter was seen, and a further attempt to determine the presence or absence of a kidney on the left was made by the use of a larger dose of the drug. Following this some visualization of the right kidney was obtained, but a shadow was not seen on the left. In spite of severe damage of the kidney, no reaction followed this large dose. However, we did not feel sure that we had absolute proof of the absence of the left kidney until after exploration.

We do not yet have definite conclusions as to the relative value of the two forms of intravenous dyes used for obtaining visualization of the urinary tract, the two drugs being iopax and skiodan.

CONCLUSIONS

The value of intravenous urography is limited in cases showing a marked degree of pyuria associated with damage of the kidney, and this group of cases comprises a large percentage (about 60) of the disorders of the urinary tract commonly seen in young patients. The evidence obtained in many such cases is inconclusive and unreliable. Its dangers are presumably slight, but must be considered. Cases should be studied carefully, and all other simpler means of diagnosis,

notably cystography, should be employed first; also, complete studies of the renal function should be made. In cases not associated with marked pyuria, intravenous urography may often do away with the need for retrograde pyelography; it may sometimes give information not obtained by retrograde pyelography. Its general use in unselected and not carefully studied cases is to be avoided, on account of both the expense and the uncertainty of the information often obtained.

It is our belief that at present the value of this method of obtaining visualization of the urinary tract is directly proportionate to the user's knowledge of diseases of the urinary tract in this age group and to his experience and skill in the technical procedures necessary for the adequate study and treatment of these conditions.

Pediatric Biographies

JOHANN FRIEDERIC ZÜCKERT
1737-1778

JOHN RUHRÄH, M.D.
BALTIMORE

Zückert was born in Berlin on Dec. 19, 1737. He started in life as an apothecary, but after four years experience began his medical studies in anatomy and medicine in 1756 at "The Charite" in his native city. Two years later he changed to the University of Frankfurt, where he was graduated in 1760 with the dissertation, "De morbis ex alieno situ partium Thoracis." Following this, he made a student journey to various German universities and finally settled in 1761 in Berlin, where he was a member of the collegium medico-chirurgicum, but poor health compelled him to withdraw from practice. He then devoted himself to writing, mostly either nonmedical or popular medical books and articles. He died on May 1, 1778.

Among his contributions were volumes on the German springs and bath resorts, on diet and on foodstuffs. Most interesting of his general works from a literary standpoint was his translation, 1784, into German of Laurence Sterne's immortal "Life and Opinions of Tristram Shandy, Gent," with the title of "Das Leben und die Meynungen des Herrn Tristram Shandy." His works on diet included at least two volumes worthy of the attention of pediatricians, especially as he taught that milk was the only diet suited to infants during the first six months of life.

Among the writings of pediatric interest are: "Von der diätetischen Erziehung der entwöhnten und erwachsenen Kinder bis in ihr mannbares Alter," or "Instruction in the Dietetic Bringing Up of Weaned and Grown-Up Children to the Age of Puberty " (256 pages), which was brought out in 1765 by August Mylius, Berlin, who also printed the others listed here. It was reprinted in 1777 and 1781. "Diät der Schwangern und Sechswochnerrinen" was published in 1767. The most interesting is "Unterricht für rechtschaffene Eltern zur diätetischen Pflege ihrer Säuglinge," published in 1764; a second edition was printed in 1771.

In the introduction Zückert said, among other things:

> The parents have in three things great influence in the welfare of the child. Firstly, the healthy, lasting and good constitution of the children depends first on the strong constitution of the parents from whom they spring; secondly, on the good nourishment, rest and uninjured stay of it while in the mother's body, and thirdly, in the sensible diet and bringing up during childhood.

The first section deals with the care of the child immediately after birth, for Zückert said that many midwives, especially in the country and small villages, often do not know what they should do. For weak infants, he advised a bath of one-third wine and two-thirds water.

D. Johann Friedrich Zückerts
der Römisch-Kayserl. Academie der Naturforscher,
und der Churmaynzischen Academie nützlicher
Wissenschaften Mitglieds

Unterricht
für rechtschaffene Eltern,
zur
diätetischen Pflege
ihrer
Säuglinge

Zweyte verbesserte Auflage.

Berlin,
verlegts August Mylius, 1771.

Title page of one of Zückert's books.

In the second section are explicit directions for binding the child, which he believed of great importance. In America, the abdominal band is the last vestige of the elaborate binds, often 30 feet long, as used in the early days of Greece and Rome, and which are still in use in Italy and other parts of Europe. Soranus of Ephesus gave elaborate directions for binding, and Zückert's are not so greatly different.

The third section deals with the nourishment, on which Zückert properly laid great stress. Mother's milk is the best as it is suited to the child and differs from the milk of animals in having more whey and less curd. He called attention to the fact that animal's milk is suited to the young of the particular species: "The child gets the milk which it sucks from its mother's breasts fresh, with all the strong, subtile and spirit parts, and the child is not only nourished but refreshed and strengthened." A statement into which much that is modern may be read with but little imagination.

Animal's milk should be warm and fresh, and if kept is liable to spoil and be harmful. Keeping an animal at hand and milking at each feeding is troublesome, although it was done in those days, and the beast deprived of the fresh air, exercise and proper fresh food gave poor milk.

Zückert called attention to the fact that medicines given to the mother or wetnurse may affect the child. If animal milk is used, this means of treating the child is lost.

There is the usual, often repeated sermon on the duty of the mother to nurse the child, but if she cannot, directions are given for the choice of a wetnurse, which had become more or less standardized and to which a great deal has not been added except the technic of the Wassermann reaction and other diagnostic aids. Zückert recommended the old nail test of Soranus for milk, which, by the way, has much to recommend it. A drop of milk on a smooth surface, if good, flows slowly; if it sticks or runs off too freely, it is not good.

Zückert gave directions for making gruels, especially of bread crumbs or wheat flour, but stated:

> Of all gruels the most healthful is that made of bread crumbs with milk or water or thin beer. They can be prepared without trouble and have a great advantage over mere gruels, which are to be regarded as unsuitable for children's food.
>
> A child must, at least for the first six months of the first year of life, have absolutely no other food except milk. After this time as when the child is weakly, after the eighth month gruel may be given but only once a day in the beginning. When it is observed that the child digests it well and has neither edema or constipation and does not get diarrhea, gruel may be given twice daily, in the morning at ten or eleven o'clock and evening about six; in addition mother's milk is given. And on this diet which is sufficient the child should be left until it is time to wean it. It should not be given the breast as often as formerly. Each dish which is added afterwards should be given in place of a nursing and at the same hour.

The fourth section treats of waking and sleeping, with many useful observations and suggestions.

The fifth section is on airing. Here are also useful suggestions, such as for weak children:

> Such children must first gradually and in such years be accustomed to raw air that their skin may become thicker and the vessels stronger and so that their bodies can bear more force and more marked changes.

On this account I do not advise the child be kept too warm in winter and the room in which it is kept made as hot as a bathroom or to place the cradle near the stove. These are the easiest ways to kill a child.

The air in the room in which the child is kept should be not too warm or too cold but temperate and free from dust and pure.

One should be careful about smoking in the room.

One should not burn strong smelling things in the room.

The sixth section treats of exercise and rest, the seventh on excretion and cleanliness. Zückert's bathing directions are taken from Tissot. Curiously enough, the most detailed account of the child's bath and toilet are by the Frenchman, Vallembert.

The eighth section deals with the diseases, and the last with certain special cautions in the care of children.

The child is frightened by the following evil usages:

1. When the nurse is infuriated and snarls at a restless child, or looks at it with an angry countenance or threatens it with the hands or stamping of the feet.

2. When the child is suddenly awakened from sleep with impetuosity.

3. When it is brought quickly out of a dark room into a bright one; or when a light is suddenly brought close to the cradle.

4. When the child cries and loud laughter and joyous noises are made to quiet it; or when one quickly appears before the child in any kind of clothing which will frighten it; or when an anxious or complaining howl is raised.

In short, any sudden, quick, unexpected sensation will frighten a child.

Temper is produced, increased and prolonged by the following things:

First by doing things which the children dislike; some parents believe that they can improve the bad temper of children by letting them have their own way but in the course of time the contrary is true for the children do not understand this, and follow their own inclinations and want their whims satisfied and when not given what they want or when things which they like are taken away, they cry and go into a temper until one is compelled to give them their wish.

Of course, the child observes this and on the next occasion when the child cannot have his way he recourses to temper for by this he has achieved his desire on previous occasions. One must, therefore, remove the desire of the thing by removing it so that when they are repressed there is no occasion to cry for it. One must avoid having among the toys in the room things which are harmful and if by accident such things fall into their hands they must be removed not by force but in a gentle manner, while diverting their attention and substituting something harmless.

Second, temper is increased by allowing them to be in the cradle without paying attention to their cries. It is natural for them to cry when tired of lying in the cradle, when hungry, or when they have pain as when the nurse cruelly neglects them, which only increases their irritation.

Third, when one permits an angry child to strike at its sisters, brothers or nurse it encourages it to do so.

Fourth, when we conduct ourselves in a gruff and unpleasant manner and without provocation throw it down or push it or strike it, it increases its badness.

It is sufficient if one earnestly threatens the child when bad for whipping does not change it because the child does not understand why it is done and soon forgets the chastisement.

News and Comment

GENERAL

Examination of School Children for Tuberculosis in Philadelphia.—The Philadelphia Health Council and Tuberculosis Committee has offered to finance tuberculosis examinations and a demonstration of methods among school children over a three year period. The council estimated that 4,000 children could be examined annually by the funds that it will make available.

Conference on Child Health for Chicago.—The Chicago area White House Conference on Child Health and Protection will be held at the Palmer House, Chicago, Oct. 30, 1931. The session will be divided into five divisions of medical service, public health education and training, child welfare and community playing. The Chicago Medical Society will head the medical service.

Wisconsin Holds Diphtheria Campaign.—The health department of Wisconsin conducted its second annual diphtheria immunization campaign during August. Toxoid and antitoxin were furnished gratis to the private physicians.

DEATHS

Dr. C. Picot, whose textbook with d'Espine appeared in 1877 and went through six editions, died recently at Geneva, Switzerland, at the age of 86. Besides his co-authorship with d'Espine, he had published many pediatric articles, and was a well known Swiss pediatrician. He helped to found the *Révue médicale de la Suisse Romande*. His death severs another link with the past, and although he had reached such advanced years, his loss is deeply regretted.

Dr. Enrico Modigliani of Rome, who was in charge of the section of contagious diseases at the Policlinico in Rome, died recently at the age of 52. During the war, when serving as a military surgeon, he contracted bronchopneumonia from which he never completely recovered. His loss will be felt in Italian pediatrics and elsewhere.

SOCIETY NEWS

University of Michigan Pediatric and Infectious Disease Society.—The next meeting of the University of Michigan Pediatric and Infectious Disease Society will be held at Ann Arbor, Mich., November 20 and 21.

Abstracts from Current Literature

Pathology, Bacteriology and Biochemistry

STUDIES ON THE MECHANISM OF WATER EXCHANGE IN THE ANIMAL ORGANISM. F. P. UNDERHILL, R. KAPSINOW and M. E. FISK, Am. J. Physiol. **95**:302, 315, 325, 330, 334, 339 and 348, 1930.

In this series of articles the results of studies on the effect of burns on the animal organism are reported. With superficial burns of the rabbit, a marked local subcutaneous edema was rapidly produced, which reached its peak in twenty-four hours and did not disappear until the end of the fifth or sixth day. This was accompanied by a marked increase in the concentration of the blood amounting at times to 70 per cent of the total blood volume with a superficial burn of one sixth of the total body surface, and probably to an even greater amount with more extensive superficial burns. The permeability of the local capillaries was greatly affected, as indicated by the passage of dyes into the edematous fluid. The reabsorption of the fluid was slow, after a brief latent period, and substances passing rather freely into the fluid showed little evidence of reabsorption, so that the increased permeability did not operate in both directions. The edematous fluid was practically blood plasma; in both the fluid and the blood serum there was a marked decrease of globulin, which can be explained apparently only as the result of the mechanism of the production of edema, and which may account for the difficulty of water retention in these circumstances. There were, however, certain marked differences in the composition of the edema fluid and the blood serum, the former showing considerably increased amounts of nonprotein nitrogen, potassium, magnesium and inorganic phosphates. Diminution of chloride content in the serum was not observed as long as the concentration of the blood was maintained within normal limits, but chloride was reduced when the concentration was maintained at a high level. The loss of substance to the edematous fluid did not affect the composition of the tissues with respect to water, ash and chloride content, indicating an apparent conservation of these substances. Throughout the entire skin there was a great increase of chloride content and of total ash, without as a rule any general augmentation of water content. While the accumulation of water in the injured area was dependent on the type of injury, this was not true of the chlorides, which were always greatly increased. The writers infer that the chlorides play a prominent part in the interchange between the blood and the injured tissue. In the case of burned skin, this local increase of chlorides persisted as long as three days; with burned muscle, considerably longer. It was found that with superficial burning, heat might penetrate into the interior cavities enough to raise the temperature appreciably, and so to induce vascular changes apparently sufficient to account for the ulcers, internal hemorrhages, etc., associated with severe burning. Little change of general body temperature was observed, and a rise after twenty-four hours appeared to be due to secondary infection. In animals in which especially severe dehydration was achieved by various methods, it was found that as a result of water deprivation or increased osmotic tension, the body tissues would lose their essential water, with death as an invariable consequence.

EGGERS, Omaha.

THE EFFECT OF VITAMIN D AND PARATHYROID HORMONE ON THE STORAGE OF CALCIUM. FERDINAND HOFF and ERNST HOMANN, Ztschr. f. d. ges. exper. Med. **74**:258, 1930.

Following the injection of 50 units of parathyroid hormone (parathormone) in cases of tetany, a biphasic reaction occurred: At first there was a rise in the blood calcium, a decrease in the alkali reserve and a lessening of the potassium-

calcium quotient; a leukocytosis with myeloid tendencies occurred and sensitivity to electrical stimulation was decreased; the symptoms of tetany disappeared. In the second phase, there was a disappearance of all these vegetative regulatory phenomena in reverse order. Parathyroid hormone and vitamin D are apparently antagonistic in their action in some respects. Experiments showed, however, that visceral calcification following the administration of vitamin D was not lessened but actually increased by the simultaneous administration of parathyroid hormone. At the same time there was decalcification of skeletal bone, at times so great as to lead to spontaneous fractures.

ZEEK, Cincinnati.

Nutrition

SPONTANEOUS SCURVY IN MONKEYS. BEATRICE F. HOWITT, Arch. Path. **11:** 574 (April) 1931.

A report is given of the spontaneous development of scurvy in a group of thirty-nine monkeys (*Macacus rhesus*) received from India. The diagnosis was made from the clinical symptoms, the x-ray pictures and the postmortem observations, all of which correspond closely to the description of experimental scurvy in monkeys given by Hart. A change of diet to one rich in vitamin C produced marked improvement within a few weeks, with subsequent complete recovery, in all of the group except those presenting advanced scurvy, which were put to death or which died during the first few days.

AUTHOR'S SUMMARY.

LOW FAT, HIGH STARCH EVAPORATED MILK FEEDING FOR THE MARASMIC BABY. JOHN HOWELL WEST, Arch. Pediat. **48:**189 (March) 1931.

The author reports favorable results in the treatment of 152 marasmic infants with a formula consisting of unsweetened evaporated milk, calcium caseinate and baked wheat flour. Directions are given for the preparation and administration of the mixture used. The results are described in a general fashion. There are no case reports.

WADDELL, University, Va.

ALIMENTARY INTOXICATION. F. ELMER JOHNSON, Arch. Pediat. **48:**194 (March) 1931.

Two cases of alimentary intoxication are reported. In one instance there was scleroderma without an abnormal condition of the blood. The other case was characterized by severe acidosis and uremic indications in the blood. Both patients recovered. The author feels that alimentary intoxication can be recognized by the toxic facies and changes in tissue turgor. In his experience scleroderma has always been followed by death, except in the case reported.

Transfusions have been of some benefit. The results in sixty-seven consecutive cases are cited. In alternate cases direct transfusions were given. The mortality in the group receiving transfusions was 64 per cent as compared with 78 per cent for those patients not given transfusions. Johnson feels that transfusions aid in overcoming the infant's inability to retain fluids. Without transfusions, the dehydration tends to persist. Transfusions were given every second day until from three to five were given.

WADDELL, University, Va.

POWDERED RIPE BANANA IN INFANT FEEDING. SIDNEY V. HAAS, Arch. Pediat. **48:** 248 (April) 1931.

The author's summary is as follows:

"The powder represents the first successful effort to powder fully ripened bananas.

"Banana powder made from fully ripened bananas removes the difficulties inherent in feeding bananas to bottle fed babies.

"The powder and method of production are described.

"In appearance, taste and value it resembles banana. It contains vitamins A, B and C in adequate amount.

"It appears probable that it transforms the intestinal flora of infants from a Gram-negative to a Gram-positive type.

"It contains an enzyme capable of hydrolyzing the starch of banana, and of converting cane sugar to invert sugar; this is destroyed by temperatures above 150 F.

"Its caloric value is 116 to 1 ounce (3 level tablespoons).

"It is well borne by infants even in the first weeks of life. In cases of intoxication it may prove to be allergic.

"There is a marked acceleration of growth in length during the first six months, approximately 20 per cent. The weight is not especially influenced.

"The infants are ruddy, firm and contented. The teeth, which do not erupt earlier than usual, are hard, white and have good enamel.

"After mixing the milk and placing on ice for 24 hours, there is no change in p_H.

"It should prove a welcome addition to the infant's dietary."

WADDELL, University, Va.

USE OF VITAMIN B IN DIETS OF INFANTS. B. R. HOOBLER, J. A. M. A. **96**:675 (Feb. 28) 1931.

One gram of wheat germ extract and 0.5 Gm. of yeast extract to the ounce of a preparation of maltose and dextrin when fed to infants caused no appreciable gain in weight over a control group. A greater growth in recumbent and stem length, however, was shown. About one out of six infants showed symptoms of rigidity, which disappeared in all but six cases, indicating that the amount of vitamin B in commercial preparations may or may not be sufficient for certain infants.

BONAR, Salt Lake City.

THE PREVENTION OF RICKETS IN PREMATURE INFANTS BY THE USE OF VIOSTEROL 100 D. E. W. MAY, J. A. M. A. **96**:1376 (April 25) 1931.

The exact dose of viosterol in the prevention of rickets is questionable, but the investigations of the author suggest a minimal dose of 30 drops and a maximal dose of 60 drops daily for premature infants.

BONAR, Salt Lake City.

ROLLED OATS AND BRAN IN A SCURVY-PRODUCING DIET AND THE NEGATIVE CONTROL TEST. E. W. SCHWARTZE, F. J. MURPHY and R. M. HANN, J. Nutrition **2**:171 (Nov.) 1929.

A diet containing 14.7 per cent of crude protein, obtained from rolled oats and bran in the ratio of 2:1, butter-fat and salts is described. This diet represents the approximate maximum amount of protein that it is possible to obtain by the ingestion of these raw materials. It is well suited to the feeding of animals. No vitamin C could be detected. The authors recommend that all commercial rolled oats, bran and dried brewers' yeast be tested by the use of animals before one assumes that no vitamin C is present.

KUGELMASS, New York.

NERVE DEGENERATIONS RESULTING FROM AVITAMINOSIS A. J. S. HUGHES, H. F. LIENHARDT and C. E. AUBEL, J. Nutrition **2**:183 (Nov.) 1929.

Lack of vitamin A in the diet of pigs, chickens and cows results in marked nervous symptoms characterized by impaired vision, incoordination and spasms. Histologic examination of the nerves of pigs in the advanced stages of avitaminosis A shows degeneration of some of the nerve bundles. This degeneration was observed in portions of the spinal cord and the optic, sciatic and femoral nerves.

KUGELMASS, New York.

COW MILK AS A SOURCE OF VITAMIN B FOR LACTATION. BARNETT SURE, Science **70**:583, 1929.

The work of Daniels, Jordan and Hutton on the requirements of vitamin B is criticized for the following reasons: There was not sufficient attempt to deplete the lactating mothers of vitamin reserves; the diet used was not complete in every respect except for the vitamin B factor; four young rats were used instead of the standard technic of using six young rats to a litter. The application of the results of these experiments to problems of infant nutrition cannot be made directly.

KUGELMASS, New York.

AVITAMINOSIS. A. PUGLIESE, Arch. ital. de biol. **81**:91, 1929.

In fasting pigeons there is always a slight fall in glycemia, the blood sugar reaching a low level which does not vary during the inanition. During avitaminosis resulting from feeding either decorticated or autoclaved rice the occurrence of hyperglycemia is not constant. The glycogen of the liver and muscles is greatly diminished; it is nearly exhausted when the animals have lost 40 per cent in weight. Whereas the inorganic phosphoric acid and the lactacidogen of the pigeon breast muscle are only somewhat diminished (9 per cent) during fasting, the situation is quite different in the pigeons suffering from B avitaminosis. In these, inorganic phosphoric acid is practically 300 per cent higher than in the fasting muscle and the lactacidogen is considerly increased also. The lactic acid of the blood, which is lower in fasting pigeons, is greatly increased in pigeons with avitaminosis as compared with that in the normal bird, and the same holds true for the muscles, though not to the same extent. The liver, on the other hand, shows a great increase in lactic acid concomitantly with the disappearance of its glycogen during avitaminosis. It is suggested that the anaerobic phase of glucide metabolism with the formation of lactic acid is not affected in avitaminosis, but the aerobic phase of glycogen resynthesis is deficient and much lactic acid is excreted.

KUGELMASS, New York.

MATERNAL FACTORS IN RACHITISM. G. MACCIOTTA, Clin. pediat. **12**: 923 (Nov.) 1930.

The author considered the possibility that apart from the postnatal factors already known to be productive of rachitic changes, there might also be certain factors relating to the maternal organism that act on the fetus during its intrauterine existence and constitute a true etiologic basis of rickets. Clinical and statistical studies in 735 rachitic children established the fact that in 45 per cent there existed, before and during pregnancy, nutritional and hygienic deficiencies in the mother from which, very probably, the fetus inherited a marked influence. Another fact that attracted attention was that 50 per cent of these infants were breast-fed.

The author therefore determined to conduct a series of experimental studies with guinea-pigs and rabbits to establish whether certain factors, acting through the maternal organism during pregnancy lead to the production of actual or potential rachitic changes in the fetus. These experiments were checked by morphologic, roentgen and histologic studies, and were carried along four lines of observation: 1. An unbalanced diet was administered to the mothers during the entire period of pregnancy. 2. The mothers of this group were kept entirely and continuously in darkness for the full period of pregnancy. 3. The mothers were diversely treated to determine the effect on the fetus of several endocrine modifications. These included extirpation of the thyroid and thymus glands, and administration of suprarenal extract (whole gland), pituitary extract and thymic hormone. 4. The mothers of this group were fed through the entire lactation period on a diet strictly devoid of antirachitic elements.

The author was able to show that while the complete elimination of phosphorus from the mother's diet produces in the fetus constant manifestations of rachitic changes, demonstrable by morphologic, roentgen and histologic evidence, the elimination of calcium alone in the diet fails to produce the same results. Mild rachitic manifestations were noted in the newly born animals whose mothers were kept in continuous darkness during the period of pregnancy, while the newly born animals whose mothers were given thyroid extract during the period of pregnancy manifested marked rachitic changes. No demonstrable rachitic developments were noted in the newly born offspring of mothers whose thyroid glands were extirpated or which had been given thymic extract, suprarenal extract and pituitary extract. The fourth group of the experiments shows that deficiencies in the maternal alimentation during the period of lactation provoke rachitic manifestations in the nursling.

The author discusses the mechanism of these manifestations and points out that cellular, metabolic and constitutional disturbances in the fetus interfere with the normal postnatal ossification. He holds that proper prophylaxis, if it shall effectively prevent the development of richets, must begin with supervision of the mother as regards nutritional and hygienic requirements from the beginning of pregnancy and actively carried on through the period of lactation.

SIGNORELLI, New Orleans.

THE RELATIONSHIP BETWEEN THE AVITAMINOSIS OF BERIBERI AND TETANY. A. SIRCA, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 236.

Observations were made on pigeons and rabbits to which a diet deficient in vitamin B was given. In pigeons, the p_H of the blood was increased from the normal average of 7.5 to 7.6, and the ionized calcium decreased from an average of 2.5 mg. to 1.4 mg. per hundred cubic centimeters as a result of the avitaminosis. In rabbits, the diet of polished rice resulted in a marked loss of body weight, an increase in the p_H of the blood from 7.55 to 7.66, a decrease in the calcium of the blood from 15.7 to 12.4 mg. per hundred cubic centimeters, a decrease in the ionized calcium of the blood from 2.25 to 1.07 mg. per hundred cubic centimeters and an increase in the potassium of the blood from 20.35 to 27.04 mg. per hundred cubic centimeters. The carbon dioxide-combining power of the blood was increased from 39.5 to 53.2 per cent by volume. The tendency toward alkalosis, diminished blood calcium and increased blood potassium is common both to parathyroid tetany and the avitaminosis of beriberi.

HIGGINS, Boston.

THE ORIGIN OF THE PROTEIN IN MILK. D. PACCHIONI, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 302.

The author gives a theoretical discussion on why casein predominates in cow's milk and lactalbumin predominates in human milk.

HIGGINS, Boston.

EXPERIMENTAL STUDY OF THE PROTEIN AND NONPROTEIN NITROGEN CONTENT OF MOTHER'S MILK. A. VENUTI, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 308.

The author made analyses of the protein and nonprotein nitrogen content of samples of milk from twenty mothers. The total nitrogen of the milk averaged 0.179 per cent (maximum, 0.210 per cent; minimum, 0.135 per cent). Of this, 0.028 per cent was nonprotein nitrogen (maximum, 0.039 per cent; minimum, 0.19 per cent). By calculation from these figures, the total protein proved to be 0.94 per cent (maximum, 1.10 per cent; minimum, 0.67 per cent). By the use of acetic acid to separate the casein from the lactalbumin, the casein content was found to be 0.49 per cent (maximum, 0.57 per cent; minimum, 0.32 per cent) and the lactalbumin 0.45 per cent (maximum, 0.54 per cent; minimum, 0.35 per cent). The

ratio of casein to lactalbumin was 1 : 0.9. Using magnesium sulphate to separate the casein, the author found the average values for casein and lactalbumin to be 0.56 per cent and 0.38 per cent, respectively, and the ratio 1 : 0.6. He feels that magnesium sulphate is not as satisfactory as acetic acid, in that the lactoglobulin is precipitated by this salt and counts in the casein fraction. Sodium chloride is an unsatisfactory reagent for the quantitative precipitation of casein.

HIGGINS, Boston.

THE PROTEIN METABOLISM IN THE ARTIFICIALLY FED CHILD. V. PINTOZZI, Scritti medici dedicati a Carlo Comba, ediz., Riv. di clin pediat., 1929, p. 353.

The author reports experiments on nitrogen balance in three bottle-fed infants, 3, 5 and 10 months of age. The constituents of the various diets were whole milk, skimmed milk, rice water, sugar, oil and plasmon. An increase in the protein of the food resulted in increased retention of nitrogen. The utilization of protein seemed independent of the quantity and quality of the fat. In such experiments, it is necessary to analyze the individual samples of food for their protein content, as the protein content of the milk varies.

HIGGINS, Boston.

CLINICAL OBSERVATIONS ON THE FAT AND NITROGEN METABOLISM IN BABIES FED WITH A SPECIAL FEEDING MIXTURE (M. A. B.). G. GUASSARDO, Scritti medici dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 382.

The author has studied the absorption of fat and retention of nitrogen by infants —both normal infants and those with nutritional disturbances—who were fed a special feeding mixture (M. A. B.). The results were very favorable for this type of feeding. M. A. B. is a type of feeding in use in the pediatric clinic at the University of Genoa (Guassardo: *Riv. di clin. pediat.* **26**:649, 1928; abstr., AM. J. DIS. CHILD. **37**: 621 [March] 1929).

HIGGINS, Boston.

EXPERIMENTAL RICKETS. P. BARBACCI, Scritti medici dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 443.

The author attempted to produce rickets in white rats by a diet very poor in phosphorus and nearly lacking in vitamins, but rich in calcium. The diet contained: rice flour, 40 per cent; egg albumin, 30 per cent; lactose, 10 per cent; sucrose, 10 per cent; calcium carbonate, 4 per cent, and sodium chloride, 6 per cent. He was unsuccessful.

Rickets was produced with MacCollum's diet no. 3143. Irradiated olive oil added to this diet protected the rats from developing rickets for as long as one month after its discontinuance; it also cured rickets already produced in rats on this diet. Also using diet no. 3143 with irradiated water, the results varied with the type of water used. A water high in organic material (well water) and a doubly distilled water showed some potency in preventing rickets, the controls showing more signs of rickets than the animals receiving the water; no effect was obtained with a water that was poor in, but not wholly free from, salts and organic material.

HIGGINS, Boston.

VALUE OF ULTRAVIOLET RAYS, CHOLESTEROL AND IRRADIATED OIL ON GROWTH AND AS CALCIUM-PHOSPHORUS FIXATIVE AGENTS. A. PAGANO, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 483.

The author made extensive observations on the effect of irradiated fats on white mice. He also used these irradiated oils on children and was much impressed by the value of inunctions as a means of obtaining the benefits of the irradiating principle.

HIGGINS, Boston.

WATER-SOLUBLE VITAMINS AND THEIR RELATION TO EACH OTHER. W. KOLLATH, Arch. f. exper. Path. u. Pharmakol. **142**:86, 1929.

Rats fed on a diet deficient in vitamin B die of symptoms characteristic of the absence of both the pellagra-preventive and the antiberiberi factors, whereas when alkaline hematin is added to the diet in sufficient quantities the symptoms are those of beriberi only. It therefore appears that alkaline hematin can function as the pellagra-preventive factor.

KUGELMASS, New York.

CLINICAL PROOF OF THE AVAILABILITY OF GRAPE SUGAR IN INFANT FEEDING. W. HERTZ, Arch. f. Kinderh. **92**:1 (Nov. 28) 1930.

The high price of maltose preparations caused Hertz to try grape sugar. He used it with either albumin milk or buttermilk in a 5 per cent concentration. Of 111 cases of dyspepsia, the results were good in 80, unchanged in 15 and poor in 16. He therefore concludes that dextrose is a satisfactory carbohydrate in infant feeding.

SANFORD, Chicago.

RICKETS: I. LONG-CONTINUED EXPERIMENTS ON THE CALCIUM AND PHOSPHORUS METABOLISM OF NORMAL AND RACHITIC INFANTS. E. ROMINGER, H. MEYER and C. BOMSKOV, Ztschr. f. d. ges. exper. Med. **73**:343, 1930.

The calcium and phosphorus balance in three normal and nine rachitic children was observed in metabolism experiments of an average duration of twenty-three days, the longest lasting for forty-seven days. Florid rickets and rickets that healed during the administration of viosterol were thus observed without interruption in the same subject. On the basis of their observations the authors divide the metabolic disturbances of rickets into four periods, the first two belonging to active rickets and the second two to healing rickets. In the first stage of the disturbance, associated with only moderate clinical and roentgenologic findings, there is a much lowered or even negative phosphorus pentoxide balance associated with a relatively good calcium monoxide balance. The ratio of excreted phosphorus pentoxide to excreted calcium monoxide is much higher than the ratio between these salts in the bones. Since the latter ratio is the same in rachitic as in normal bones a part of the excreted phosphorus must be derived from the rest of the body. In the second stage, when rickets is clinically and roentgenographically most pronounced, the authors find the phosphorus pentoxide and calcium monoxide balance either slightly positive or slightly negative. In the third stage, that of early healing, there is a very high positive phosphorus pentoxide balance, much higher than the calcium monoxide balance. The retained phosphorus penoxide is in much greater proportion to the calcium monoxide retained than the ratio of phosphorus pentoxide to calcium monoxide in the bones. This period is therefore the "positive phase" of the first period. In the fourth stage the calcium monoxide retention reaches approximately the level of the phosphorus pentoxide retention and both are above normal. Previous metabolic investigations of rickets have covered brief periods, describing as "florid rickets" any of the first three phases of the disturbance, which accounts for the contradictory results reported.

It is concluded that rickets is primarily a disturbance of the phosphorus metabolism, most marked in the first stage. The disease of the bones follows, and the calcium disturbance occurs secondarily. The disturbed ossification, hypophosphatemia, slowed metabolism, acidosis and secondary symptoms of rickets are explained by the phosphorus impoverishment of the entire organism. Tetany develops especially when there is sudden healing in the first stage of rickets. In the phosphorus-impoverished body there is suddenly excessive phosphorus retention, causing hyperphosphatemia. Since the calcium retention cannot keep pace with the phosphorus retention, and ossification is stimulated, calcium is withdrawn from the rest of the body to be deposited in the bones. The organism, including the nervous system, which is already low in calcium, gives up more calcium, with resulting manifestations of tetany.

CRANE, Chicago.

ALIMENTARY FEVER: IV. SODIUM CHLORIDE FEVER. H. FINKELSTEIN and E. WEIL, Ztschr. f. Kinderh. **50**:259 (Nov.) 1930.

Sodium chloride fever occurs not only as a concentration fever after a single dose of hypertonic solution, but also after continued doses of a physiologic solution. Moreover, there is a sodium chloride fever with water deprivation after the administration of a physiologic solution in quantities of only 50 cc. per kilogram; in such small doses a physiologic solution of half strength can produce fever. The same is true for Ringer's solution. Sodium chloride concentration fever appears in from two to six hours; the more concentrated the solution, the earlier and more frequently it appears. The fever caused by physiologic solution and that caused by sodium chloride with water deprivation begin, with rare exceptions, in eight hours, at the earliest.

There is no basis for the theory that the sodium chloride of the blood has a pyrotogenous action. The fever is much more a result of the hydropigenous characteristic of sodium chloride by virtue of which it draws out so much water that the metabolism reaches a condition of water deficiency.

The processes that are released by the taking of sodium chloride are to be looked on as a defense mechanism to a foreign substance, which calls forth by its powerful affinity for water a reversal of the decrease in pressure, by which the water is drawn from the places of greatest need to territories of less need: the kidneys, connective tissue and muscle. This defense mechanism consists of diminishing the fall in pressure by simultaneous excretion of sodium chloride and dilution of the residue as soon as possible. If this succeeds in sufficient degree and quickly enough, the reaction is without fever; if not, there is fever. Whether it succeeds depends on the size of the dose of sodium chloride, on the quantity of water given simultaneously and on the individual characteristics of the patient.

With excessive concentration of salt and little water, with such a ratio as 3:100, a sufficient diminution of the difference in pressure cannot be attained. After doses of 3 Gm. and under, on the other hand, the diminution is possible with a frequency inversely proportional to the size of the dose. Here the individual course of the reaction is decisive. If it is coordinated, that is, if excretion and dilution run steadily and uniformly, helping one another, there is no fever, obviously because under these conditions enough water can still be diverted for the principal places of normal need. On the other hand, if it is not coordinated, that is, if there is a sudden quiet excretion of water through the intestines or kidneys, while a noteworthy quantity of sodium chloride is retained, fever appears, obviously because the fraction previously flowing into metabolism is immediately deflected in another direction, while the stabilizing of the water economy reached through the retained sodium chloride makes an immediate substitution impossible. One many see in this increased tendency to sodium chloride fever the expression of an excessive sensitiveness of the factors regulating water economy, which is grounded deep in the constitution. The analogy to certain tendencies in protein fever is obvious.

In the same way the differences are explained in physiologic solution of sodium chloride. Here there is a hypertonia in every case through superimposed salt, and a stabilizing and lack of mobility of water economy; there is no fever in coordinated continuation of the defense mechanism, and there is fever in time and causal relation to acute variations, which are produced by suddenly increased diuresis, diarrhea or transient interruption of the supply of water.

In water deprivation, the speed of excretion of salt is decisive. With quick excretion the water in the body remains sufficiently mobile to spare the metabolism; with slow excretion this is prevented by hypertonic stabilization. The basic resemblance to urea fever, in which there is similarly a scanty supply of water, obtrudes itself; with urea, there is water abstraction through the excessive flow of urine; with sodium chloride, abstraction by migration and fixation of water in the tissues.

The perspiration is mildly diminished in salt concentration without fever; the covering of the increased water need for retention and urine is thereby relieved.

With fever, the perspiration is increased, whereby the excess at most comes from the storage of urinary water or from diminished retention, but under certain circumstances it is further increased by a quota taken from the tissues. In the administration of physiologic solution of sodium chloride, on the whole, as much water is excreted without fever as in the addition of the same amount of salt-free fluid, but in some cases somewhat more goes out extrarenally, for a reason not known. With fever, another fraction is added, which is taken from the tissues by a mere shift in the manner of excretion. In general, there is less perspiration with fever absent and more perspiration with fever present, corresponding to conditions established elsewhere in fever, and in agreement with the results of research on perspiration in protein fever. An increase of temperature coincident with a diminution of perspiration—as would be expected in salt fever—from temperature stasis was never observed.

EMERSON, Boston.

ALIMENTARY FEVER: V. THE PHARMACOLOGY OF PROTEIN AND OF SALT FEVER. H. FINKELSTEIN and E. WEIL, Ztschr. f. Kinderh. **50**:288 (Nov.) 1930.

Differentiation between real fever and hyperthermia on the basis of the reaction to antipyrine or amidopyrine meets the difficulty that increases in temperature caused by toxins or infections cannot always be reduced by antipyretics. The observations of Finkelstein and Weil show such a far-reaching capacity not only of protein fever, but, also, in contradiction to Orosz, of sodium chloride fever for being influenced by the drugs mentioned that it is to be concluded that there is an identity rather than a difference in the usual conditions of fever.

EMERSON, Boston.

PROGNOSIS IN SEVERE RACHITIC DEFORMITIES AND THE BODY STRUCTURE OF CHILDREN AT ONE TIME RACHITIC. ALTERTHUM, Ztschr. f. Kinderh. **50**: 296 (Nov.) 1930.

Twenty-four children with severe general rickets who were born during the war and during post-war years and who were treated conservatively for an average of 8.8 months in the wards of the Kinderheilanstalt of Berlin between 1920 and 1924 were followed up in 1927. The study showed marked retrogression of the visible deformities of the head as well as of those of the thorax and extremities. Measurements taken, according to anthropometric requirements, of the circumference of the head and chest, body length, sitting height and breadth of shoulders and hips showed, in agreement with the somatoscopic observations in almost all the children: a deficit in length corresponding, on the average, to a half year; an overlength in sitting height, and an oversize in the circumference of the head and chest and in the breadth of the shoulders and hips. The body structure of children who have had severe rickets at one time appears, accordingly, to show characteristic changes at the ages of about from 5 to 10 years.

EMERSON, Boston.

ACID-BASE ECONOMY: THE MEANING OF ORGANIC ACIDS IN URINE DURING HUNGER. H. FASOLD, Ztschr. f. Kinderh. **50**:367 (Nov.) 1930.

The excretion of organic acids in the urine during hunger can be brought almost to the point of disappearance by the exhibition of free hydrochloric acid. On the other hand, the excretion is extraordinarily increased by alkali. Organic acids in the urine are excreted mostly (on the average, 81 per cent) bound to base. During hunger no change in the size of the bound fraction occurs. Accordingly, during hunger the excretion of free acid valences by organic acids is small, and excretion of acids occurs almost exclusively as phosphate. It is concluded

from the behavior of organic acids after exhibition of acids and alkalis that organic acids are not always to be considered as signs of a slowing of metabolism.

EMERSON, Boston.

NUTRITION AND GROWTH VALUES OF ZWIEBACK. I. ABELIN, Ztschr. f. Kinderh. **50**:465, 1930.

The stimulative action of zwieback on growth is much weakened by baking. The protein bodies of flour are poor in some biologically significant amino-acids and diamino-acids. It seems that certain amino-acids favoring growth, e. g., lysin, cannot withstand the action of heat, and this assumption is supported by the following experiments: (*a*) The peroral feeding of very small quantities of lysin (20 mg.) daily favored growth in rats otherwise fed exclusively on zwieback. With the omission of lysin, growth became stationary. (*b*) Lysin added to dough and subjected to heat was inactive. The special significance of the baking process as a disturbing factor is that by the avoidance of baking and by the use of the Einbach method (i. e., no preliminary heating stage) the average gain in weight is much better. This injury to a biologically important compound is more important than a contingent deficiency in vitamins. In spite of the daily addition of 1 cc. of fresh brewers' yeast, of lecithin or of from 2 to 5 cc. of fresh milk, rats did not show a good increase in weight with zwieback. The substances rich in vitamins often given in zwieback, such as butter or whole milk, did not noticeably better the growth. Feeding young rats for weeks with bread zwieback caused them to have an extensive falling out of hair, which was due among other things to a thyrogenous injury. By giving very small amounts of dried thyroid gland, the loss of the hair could be avoided and its growth stimulated. Cystin also favors the growth of hair.

EMERSON, Boston.

THE PHYSIOLOGY OF DIGESTION IN INFANTS: XII. THE NATURE OF THE SOLUTIONS OF CALCIUM AND PHOSPHORUS IN CHYME. O. BUDDE, Ztschr. f. Kinderh. **50**:482, 1930.

Budde studied the influence of the digestive processes on the ultrafiltrability of the calcium and phosphorus of milk and obtained the following results: Seventy per cent of the total calcium of human milk, and from 20 to 30 per cent of cow's milk was ultrafiltrable, the corresponding figures for phosphorus being 36 and 52 per cent; acidification and proteolysis increased the amount; rennet whey did not contain more than raw milk; lipolysis diminished considerably the calcium fraction by the formation of calcium soap; the bile acids did not affect the amount as concerns calcium or the formation of calcium soap; neither oxidation nor desaturation of fatty acids changed the formation of calcium soaps; in the action of duodenal juice, the precipitating effect of lipolysis was greater than the dissolving effect of proteolysis; the result was a diminution of ultrafiltrable calcium. Boiling, but not pasteurization (for thirty minutes at 70 C.) diminished the amount distinctly.

EMERSON, Boston.

RELATION OF THE PREEN GLAND (GLANDULA UROPYGIALIS) OF BIRDS TO RICKETS. H. C. HOU, Chinese J. Physiol. **3**:171, 1929.

Removal of the preen glands from adult birds produces in some (fowl and duck) a marked disturbance of the plumage and an impairment of general health and in others (pigeons) only a slight disturbance of the plumage. Removal of the preen glands from young rachitic or normal fowls produces permanent rickets in spite of subsequent normal feeding, environment and sunshine. Occlusion of the opening of the preen glands of ducks causes some disturbance of plumage and loss of weight. The possible relationship between the sebaceous secretion and the formation of the antirachitic vitamin is discussed.

[ARCH. PATH.].

Prenatal Conditions

CONGENITAL DILATATION OF URETER. HUGH THURSFIELD, Proc. Roy. Soc. Med. **24**:561 (March) 1931.

A girl, aged 3 years, had been subject to attacks of fever accompanied by the passage of thick muddy urine since 3 months of age. Recently the urine had been continuously turbid and the child had complained of abdominal pain. The urine contained blood and pus. Cultures showed *Bacillus coli* and *Staphylococcus albus*. Roentgen examination after the injection of iopax intravenously showed an almost normal renal pelvis and a greatly dilated ureter on the left side. The right side was not clearly visualized. The iopax was held up in the left ureter to such an extent as to be still clearly visible outlining the ureter three hours after the injection. The report is accompanied by pictures of pyelograms showing dilated ureters with a ureteral catheter in situ.

WILLIAMSON, New Orleans.

CONGENITAL DEFORMITIES WITH STIPPLED EPIPHYSES AND CONGENITAL CATARACT. HUGH THURSFIELD, Proc. Roy. Soc. Med. **24**:564 (March) 1931.

A patient, aged 11 months, was noticed not to be growing properly from the time of birth. The maximum circumference of the head was 19 inches (48.2 cm.). The palate was high and arched. The skin of the face and head was dry and scaly. Double congenital cataract was present. The arms and legs were short as in achondroplasia. There was limitation of movement at the hip joints, extension, external rotation and abduction being difficult. Roentgen examination showed: 1. The humeri were shaped like mushrooms at both ends. 2. The epiphyses were stippled and the shafts short. 3. The femoral necks were abnormally ossified and the hips appeared to be congenitally dislocated. 4. Ossification in the necks of the ribs and in the vertebrae was abnormal, and there was stippling of the epiphyses of the pelvis. The report is accompanied by roentgenograms of both the upper and the lower limbs.

WILLIAMSON, New Orleans.

A RARE CASE OF MALFORMATION OF THE HEART: LACK OF THE PULMONARY ARTERY AND OF THE DUCTUS ARTERIOSUS. A. MALAGODI, Scritti medici dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 375.

The author presents a case of congenital malformation of the heart in a child who lived but five months. Cyanosis, occasional coughing and dyspnea, hippocratic fingers and cardiac enlargement were present from birth. At no time was a murmur heard. The sounds were weak and the pulmonic second sound absent. At necropsy, the findings were: (1) absence of the pulmonary artery, (2) absence of a ductus arteriosus, (3) a patent interventricular septum, (4) a patent foramen ovale, (5) enlargement of the right ventricle and (6) dilatation of the aortic bulb. The author was unable to ascertain how the blood reached the lungs in this case. In similar cases, the bronchial arteries, the coronary arteries, the esophageal artery or branches from the thoracic or abdominal aorta have assumed this function.

HIGGINS, Boston.

Acute Infections

RAT BITE OF THE EYELIDS RESULTING IN RAT-BITE FEVER. C. M. SWAB, Am. J. Ophth. **13**:884 (Oct.) 1930.

A Negro boy, aged 6, was bitten on the left eyelid by a rat. Two weeks later the eye and the left side of the face became so swollen that the eye could not be opened. There was no pain but some fever. Examination showed an edematous swelling of the left lids. The temperature was 100.4 F. At the outer canthus were two depressed marks in the skin of the lids, one in each lid; that in the upper lid resembled the impression of a rat's tooth and was fully healed.

The wound on the lower lid was covered by a rather heavy crust, which on removal disclosed a fully healed lesion of the skin. The conjunctiva was somewhat edematous and injected, but all of the other ocular structures were normal. There was a marked, firm swelling of the anterior cervical lymph glands on the left side. A blood count showed 4,800,000 erythrocytes, 10,500 leukocytes, 70 per cent polymorphonuclears and 85 per cent hemoglobin. A Wassermann test was not made. The patient improved under treatment with neoarsphenamine, and was discharged at the end of sixteen days with only slight edema of the lids.

REESE.

CHRONIC DIPHTHERIA ORIGINATING IN AND LIMITED TO THE TRACHEA AND BRONCHI. A. E. BULSON and E. L. BULSON, Ann. Otol., Rhin. & Laryng. **39**:1039 (Dec.) 1930.

Chronic diphtheria originating in the trachea and limited to that region throughout its course is an exceedingly rare condition. Such a case is reported by the authors who, after a careful search of the literature, find no identical condition described during the past fifteen years. The patient, a housewife, aged 67, was observed carefully for fifteen months; at no time was there a membrane above the vocal cords, and diphtheria bacilli could not be demonstrated by smear or culture from the tissues above the larynx. With the laryngoscopic mirror some of the membrane could be seen immediately beneath the cords. Fragments of this membrane were coughed up from time to time, and it was from this that the diagnosis was made, as diphtheria bacilli were found in every piece examined. The patient was practically free from symptoms, except for an occasional cough when small pieces of the membrane were coughed up without difficulty. She had had influenza ten years previously and her general health had been poor since then, but she was never confined to bed. General physical examination gave essential negative results. Two doses of antitoxin, consisting of 40,000 units each, were administered without results. Extensive autogenous vaccine treatment was instituted, without improvement, except that slightly larger pieces of membrane were coughed out. Injections of toxin-antitoxin were given, which also proved ineffective. At the time of the report the patient continued in fairly good general health, without symptoms except that she coughed up a small amount of membrane two or three times a month. A bronchoscopic examination was never made, as the patient objected to the procedure, but the authors feel that this method offers by far the best chance of relieving the condition.

SNAPP, Grand Rapids, Mich. [ARCH. OTOLARYNG.]

ENCEPHALITIS AND ENCEPHALOMYELITIS IN MEASLES. ARMANDO FERRARO and I. H. SCHEFFER, Arch. Neurol. & Psychiat. **25**:748 (April) 1931.

Histologically, postmeasles encephalomyelitis shows a perivascular proliferation together with a perivascular demyelinization. Frequently there are thrombi and vascular changes, swelling or hyperplasia of the endothelium. These changes point to a participation of the vascular system in the process. This fact associated with the one that most of the lesions are perivascular in topography justifies the hypothesis that whatever noxious agent is the cause of the lesions, this agent is carried from the blood vessels to the surrounding tissue and is favored by an abnormal permeability of the protective wall and stasis of the venous system. Six cases are reported.

BEVERLY, Chicago.

SALICYLIC ACID FRUIT IN THE PREVENTION AND TREATMENT OF RHEUMATISM IN CHILDREN. J. EPSTEIN, Arch. Pediat. **48**:73 (Feb.) 1931.

The author advocates the use of salicylic acid fruit for the prevention of rheumatism. The article lacks scientific accuracy and adds nothing to the knowledge of the subject.

WADDELL, University, Va.

MILKBORNE OUTBREAKS IN NEW YORK STATE. PAUL B. BROOKS, New York State J. Med. **30**:1418 (Dec. 1) 1930.

Ninety-one milk-borne outbreaks of communicable disease occurred in New York State between 1917 and 1929, inclusive. In only two instances had the milk been pasteurized. Eighty per cent of the milk sold in the cities is pasteurized. The number of cases in the series reported was 2,511. Seventy per cent of the total of ninety-one outbreaks were of typhoid and paratyphoid. While the outbreaks of septic sore throat were few, the number of cases in the individual outbreaks was large. There were twelve outbreaks of milk-borne scarlet fever, with 409 cases. Contamination occurred at the farms in 80 per cent of the ninety-one outbreaks. Carriers on the farm accounted for forty-nine outbreaks of typhoid. Cities were affected in 30 per cent and rural areas in 70 per cent of the instances reported.

AIKMAN, Rochester, N. Y.

CURRENT PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES. Pub. Health Rep. **46**:388 (Feb. 20) 1931.

The incidence of influenza and scarlet fever showed an increase for the period from Dec. 28, 1930, to Jan. 31, 1931. The rate of poliomyelitis, smallpox, measles, diphtheria and meningitis decreased markedly. The mortality averaged 14.5 per cent as compared with 13.5 per cent for the same period last year.

SANFORD, Chicago.

THE BLOOD CONSTITUENTS IN ACUTE RHEUMATISM BEFORE AND AFTER SALICYLATE TREATMENT. E. M. HICKMANS and S. H. EDGAR, Arch. Dis. Childhood **5**:387 (Dec.) 1930.

Before treatment with salicylates three cases of acute rheumatism showed somewhat higher figures for blood urea, nonprotein nitrogen and uric acid than those obtained for normal children of the same age.

Although salicylates increase the output of total nitrogen, urea and uric acid through the kidneys, and the blood uric acid is thereby decreased, the examination of a large number of patients with rheumatism during treatment indicates that the blood urea and nonprotein nitrogen content still remain slightly higher than normal during treatment; since the phosphorus content of the blood is also slightly higher than normal, it would seem that in rheumatism the kidney is unable to deal adequately with these two products. That this inability is due to some impairment of renal efficiency as a result of the acute rheumatic infection rather than to the salicylate taken is suggested by the fact that the blood urea of normal children was not found to be increased by similar quantities of salicylates. On the other hand, considering that the level of blood urea rises after salicylates are given, it is possible that salicylates may increase to some extent the degree of renal inefficiency.

KELLY, Milwaukee.

THE IMMUNOLOGY OF SCARLET FEVER: AN UNUSUAL CASE. W. R. F. COLLIS, Arch. Dis. Childhood **5**:441 (Dec.) 1930.

In a boy, 26 months of age, with empyema (pneumococcus) a scarlatiniform rash developed which faded in twenty-four hours. The Dick test was negative. Twenty-four days after the rash, the urine contained albumin, blood, pus and casts. The patient's condition grew worse, and he died in ten days. A second Dick test proved negative. At postmortem examination a pure culture of hemolytic streptococcus was grown from blood taken from the heart. This organism was isolated from the spleen and the pericardial and peritoneal fluids. The microorganism was classified as belonging to type II scarlatinal streptococcus.

This case showed only transient rash and none of the graver toxic symptoms of the scarlet fever syndrome, yet the patient succumbed to a specific hemolytic

streptococcus which is generally accepted as the cause of the disease. The Dick test was negative at a time when, under ordinary conditions, one would have expected it to be positive. The author concludes that the case affords additional evidence that scarlet fever is not a simple toxin-antitoxin phenomenon, and that the Dick test should be regarded merely as an indication of the presence of antitoxin in the patient's serum and not as a guide to the general immunity of the patient to the specific streptococcus of the disease.

KELLY, Milwaukee.

PURPURA HÆMORRHAGICA COMPLICATING SCARLET FEVER. CHARLES R. BOX and RICHARD MASSINGHAM, Lancet **1**:295 (Feb. 7) 1931.

In a boy, aged 10 years, suffering with scarlet fever, petechiae were noticed on the abdomen on the tenth day of the disease. On the following day they had spread all over the body, and on the twelfth day profuse epistaxis, bloody urine, vomiting of blood and melena and abdominal colic were noted. He was admitted in a critical condition with a profuse purpuric eruption covering the trunk, limbs and face, the larger spots measuring 3 cm. Blood oozed from the nose and posterior nasopharynx.

Transfusion of blood was postponed while a trial was made of the free administration of liver extract. A quantity equivalent to 1 pound (453 Gm.) a day was administered for a fortnight. Within twelve hours there was obvious amelioration of the hemorrhagic symptoms. The epistaxis slowly ceased, and after forty-eight hours there was no hematuria. The purpuric rash slowly faded.

On the first day the blood count showed: hemoglobin, 80 per cent; red blood cells, 3,900,000; white blood cells, 80,400, and polymorphonuclears, 88 per cent; no platelet count was made. On the second day the platelet count was reported as 0 in 4,000 cells; on the seventh day, 47,300; on the fourteenth day, 93,840, and three weeks later, 380,000.

The authors state: "There is now plenty of evidence that the administration of liver causes a rapid and progressive increase in the number of blood platelets and it appears to be the most efficient means in this direction which we possess."

LANGMANN, New York.

DOUBLE HEMIPLEGIA FOLLOWING DIPHTHERIA. C. WORSTER-DROUGHT and T. R. HILL, Proc. Roy. Soc. Med. **24**:320 (Jan.) 1931.

A patient, aged 13 years, was admitted to the hospital suffering from a severe attack of diphtheria with signs of cardiac weakness which had been present about ten days. Nineteen days after the onset the patient showed paresis of the right side of the face and arm followed by partial motor aphasia and right hemiplegia. Twenty-five days after the onset a generalized epileptiform attack occurred, after which the legs were held in flexion and the patient became incontinent. The cerebrospinal fluid was negative. On the patient's admission to the hospital a double hemiplegia was found.

WILLIAMSON, New Orleans.

"TYPHOID FEVER": A CLINICAL STUDY OF TWO HUNDRED AND FIFTY-SIX CASES IN PEIPING. S. H. ZIA, Nat. M. J. China **14**:105 (April) 1928.

A study of the distribution, symptomatology, diagnosis and complications of 256 cases of typhoid fever in Peiping Union Medical College is presented.

LUCAS, San Francisco.

POSTDIPHTHERITIC LEVOGYRIC PARALYSIS OF THE EYES. JEAN SÉDAN, Rev. d'oto-neuro-opht. **8**:728 (Nov.) 1930.

This case report is one of a group of sixteen, published previously. It concerns a girl, aged 14, with obstinate postdiphtheritic paralysis of lateral

vision. She was unable to turn the eyes to the left, but convergence was not affected. There was slight deviation to the right, equal in the two eyes; diplopia was not present. General physical and neurologic examinations gave negative results. The Bordet Wassermann reaction was negative. The patient's mother had had five other children and no abortions. Questioning elicited the history of a previous, slight case of angina, and a culture from the throat showed the presence of Löffler's bacillus.

Energetic serum therapy was instituted without other effect than the production of rheumatic pains and cutaneous reactions. Improvement began after the use of 390 cc. of serum. The injections were stopped for twelve days, and the improvement ceased. The serum was again given in doses of 20 cc., three times a week, until the patient had received a total of 620 cc., when ocular movements were restored to normal.

If the Wassermann reaction had been positive in this patient, and persistent search had not been made for the causal factor, antisyphilitic treatment might have been given, which would have resulted in the loss of opportunity to make the true diagnosis, since the throat meanwhile would probably have become free from the diphtheria organism.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

CAN MEASLES ATTACK THE SAME CHILD MORE THAN ONCE? M. DA ROCHA, Arch. de pediat., Rio de Janeiro **27**:58 (Dec.) 1930.

Writers on this subject distinguish between recidivation and a "second infection." Recidivation, occurring from fifteen to sixty days after the first infection, although rare, is found more frequently than a second infection. Authenticated cases of both are to be found in the literature. Variations occur in the degree to which immunity can be acquired by an individual and they depend on his ability to form antibodies; a defect in this respect may be hereditary.

OLIVER, Columbus, Ohio.

STUDY OF POSTVACCINAL ENCEPHALITIS. P. WIDOWITZ, Arch. f. Kinderh. **92**: 81 (Dec. 19) 1930.

Thirteen cases of encephalitis were studied of which two followed vaccination. The types fell into three groups: the hypertonic, spastic-athetosic and choreiform. In the postvaccinal cases the incubation time was from nine to twelve days. The etiology is discussed as to whether the cause of postvaccinal encephalitis is the vaccine itself or the setting up of antibodies that make the body more permeable to already existing organisms.

SANFORD, Chicago.

A POLYMORPHIC EXANTHEM AS A COMPLICATION OF AN ACCIDENTAL VACCINATION. S. SEREFIS, Dermat. Ztschr. **60**:260 (Feb.) 1931.

An accidental vaccination occurred in the genital region of a woman, aged 38, who had been successfully vaccinated twice before, the last time twelve years previously. During the course of the vaccinia, a generalized eruption resembling erythema multiforme developed, and regressed in four days. This complication is rare even with a first vaccination and has not been previously reported in a patient who has already had one successful vaccination.

TAUSSIG, San Francisco [ARCH. DERMAT. & SYPH.]

THE SERUM TREATMENT FOR SCARLET FEVER IN PRACTICE. FELIX SCHLEISSNER, Kinderärztl. Praxis **1**:65 (Oct.) 1930.

Schleissner gives high praise to scarlet fever serum in combating the toxic symptoms of that disease, but he is pessimistic about its effect in preventing complications. The dose is from 10 to 20 cc. of the concentrated serum given, if pos-

sible, during the first twenty-four hours. In severe cases a higher dosage may be given, and if caution is exercised, the serum may be used intravenously. The anaphylactic reactions are severe. Passive immunization should seldom be employed.

HUENEKENS, Minneapolis.

DIFFICULTIES IN THE DIAGNOSIS OF DIPHTHERIA. PAUL WIDOWITZ, Kinderärztl. Praxis **2**:49 (Feb.) 1931.

The administration of antitoxin during the first twenty-four hours of diphtheria is 100 per cent effective. The author reviews the difficulties that prevent this early diagnosis.

HUENEKENS, Minneapolis.

TREATMENT FOR MALIGNANT DIPHTHERIA. E. KUNDRATITZ, Med. Klin. **26**:1289 (Aug. 29) 1930.

In the malignant form of diphtheria the main reason for failure of the antitoxin is that the toxin is strongly anchored to the tissues. According to the author's reasoning, one can liberate the toxin by increasing the circulation of fluid in the tissues. In experiments on animals, theophyllin seemed to increase the effectiveness of antitoxin considerably. Guinea-pigs were given subcutaneous injections of 1½ lethal doses of toxin. After varying lengths of time, the first half of the toxin was given; then during the next twenty-four hours a dose of theophyllin was given, followed by the rest of the antitoxin. The author thinks that it is important to inject some serum before the theophyllin is given, because otherwise the liberated toxin might have dangerous effects. Experiments on animals showed a decreased mortality as a result of the combined injections of theophyllin and antitoxin. The encouraging results of experiments on animals induced the author to try out the method in children with malignant diphtheria. His method was as follows: At admission from 10,000 to 20,000 units was given intravenously. A half hour later theophyllin was injected subcutaneously in a dosage of from 0.5 to 1 cc. After another half hour from 10,000 to 20,000 antitoxin units was injected intramuscularly. Six hours later theophyllin was given, and in a half hour 10,000 units of antitoxin. Twelve hours later theophyllin and 10,000 antitoxin units were given. In some cases another similar dose was given in twenty-four hours. The results are encouraging, as the mortality among the children treated by this method was much lower than among those who received other treatment. The method is of limited value in advanced cases, but it deserves attention in cases in which serum treatment alone is almost useless.

ZENTAY, St. Louis.

ACTIVE IMMUNIZATION AGAINST DIPHTHERIA WITH SUBNEUTRAL TOXIN MIXTURES. R. OTTO and G. BLUMENTHAL, Med. Klin. **26**:1886 (Dec. 19) 1930.

A subneutral toxin-antitoxin mixture 1:10 was used in immunizing guinea-pigs. The results show that the antitoxin content of the blood of these animals was higher than in other series of experiments in which neutral mixtures were used. The mixture lost a great deal of its immunizing effect after two months of storage. Guinea-pigs are particularly good for testing the toxic and immunizing properties of the different preparations.

ZENTAY, St. Louis.

POSTVACCINAL ENCEPHALITIS AND GENERALIZED VACCINIA. M. WEICHSEL, Monatschr. f. Kinderh. **49**:28 (Jan.) 1931.

In a child, aged 1½ years, a generalized vaccinia and encephalitis developed nineteen days following vaccination. The case is used to offer confirmation of Leiner's theory that the encephalitis following vaccination may be due to cerebral metastases.

GERSTLEY, Chicago.

THE PATHOMORPHOSIS OF DIPHTHERIA AS ILLUSTRATED IN COLOGNE, 1925-1930. W. VON FREY and F. THOENES, Monatschr. f. Kinderh. **49**:33 (Jan.) 1931.

A study was made of diphtheria in the city of Cologne. Basing their conclusions on the statistics of the city of Cologne since 1907 and on the statistics of the Kölner Kinderklinik for the last five years, the authors state that diphtheria has changed in type in the last year and a half. There is unquestionably a diminution in the number of laryngeal stenoses and an increase in the toxic forms of pharyngeal diphtheria with severe cardiac and nervous complications. The general mortality has not increased in proportion to the number of cases. Deaths from myocarditis in the last year and a half have shown a definite increase with virtual disappearance of the fatal diphtheritic stenoses. Complications have been more frequent.

On the basis of their material they have the clinical impression that patients with severely toxic diphtheria were not aided by large doses of serum. The failure of the serum may be explained by the increased virulence of the disease and the fact that the patients, not being aware of the increased virulence, did not come for treatment soon enough.

GERSTLEY, Chicago.

DIPHTHERIA PROPHYLAXIS BY MEANS OF LÖWENSTEIN'S OINTMENT. M. STRANSKY, Wien. klin. Wchnschr. **43**:1316 (Oct. 23) 1930.

Stransky first enumerates the disadvantages of immunization against diphtheria by means of injections. Because of the dangers involved in these methods he himself employed Löwenstein's prophylactic ointment, which contains diphtheria bacilli that have been detoxicated by a formaldehyde solution. In Vienna about 10,000 children were immunized against diphtheria by means of inunctions with Löwenstein's ointment. On the basis of his observations the author comes to the conclusion that Löwenstein's ointment is an effective prophylactic against diphtheria. The main advantages are its harmlessness, its simple technic and especially its effectiveness.

A. F. ABT, Chicago.

THE CHLORINE CONTENT OF THE BLOOD OF CHILDREN WITH SCARLET FEVER OR DIPHTHERIA. A. A. MARKOWA, Ztschr. f. Kinderh. **50**:496, 1930.

The chlorine content of the blood of children is lowered at the onset of scarlet fever or diphtheria, and the diminution is directly dependent on the severity of the disease, the cause being the disease itself, i. e., the specific infection and not the complications.

EMERSON, Boston.

MALIGNANT DIPHTHERIA. F. SZIRMAI, Orvosi hetil. **74**:807 and 842, 1930.

The author analyzes the epidemiologic and clinical phenomena observed during the last epidemic of diphtheria in Budapest. He comes to the following conclusions: 1. The pathogenic action of the diphtheria bacillus manifests itself in two directions; (*a*) The production of pseudomembranes is a local infectious process that cannot manifest itself if the organism has a good supply of antitoxin. It can easily be influenced by injections of antitoxin. (*b*) Lesions due to toxin develop more slowly and can hardly be influenced by injections of antitoxin.

2. The production of pseudomembranes predominates in laryngeal croup. General toxicity is more pronounced in diphtheria of the throat, particularly in the malignant form. The average case of diphtheria of the throat shows general toxicity only in case of neglect and then, as a rule, takes the form of postdiphtheritic paralysis.

3. The virulence of diphtheria bacilli shows a great variability in both the production of pseudomembranes and toxicity. This probably explains the widely different observations in various epidemics.

4. Malignant diphtheria is characterized by delayed development of toxicity. The reason for this is probably in the disproportion between the production of toxin and the supply of antitoxin in the blood. It appears that the toxin production of diphtheria bacilli was markedly increased in the last epidemic.

5. In malignant diphtheria there is a rather intensive toxic effect also at the site of the primary lesion, probably due to the massive production of toxin.

6. Mixed infections play only a secondary part in the development of malignant diphtheria. The effectiveness of the antitoxin has not changed. The good results in the early period of the use of antitoxin probably were due to the fact that in the epidemic of the nineties the production of pseudomembranes was rather the dominant feature of diphtheria while toxicity was less marked.

7. In malignant diphtheria the specific treatment has to be started on the first day. On the second day it is too late. In laryngeal croup the chief cause of death is bronchial pneumonia which is not due directly to diphtheria bacilli.

8. In view of the recent increase of malignant diphtheria and the ineffectiveness of serum in such cases, the use of preventive measures must become more general.

ZENTAY, St. Louis.

PARATHYROID EOSINOPHILIA IN CHILDREN WITH DIPHTHERIA. G. LENART and E. LEDERER, Orvosi hetil. **74**:1091, 1930.

The behavior of eosinophils was studied in children in the second week after diphtheria. One unit of parathyroid extract-Collip per kilogram of body weight was injected subcutaneously. The eosinophils were counted five hours, and twenty-four hours, after the injection. For staining, Dunger's solution was mixed in the pipet used for diluting the blood. The results show that in children who have had diphtheria the eosinophils show somewhat different behavior from those in normal children. In normal children the eosinophil count shows a decided decrease five or six hours after the injection, conditions returning to normal in about twenty-four hours. In children who have recently had diphtheria there is an increase of eosinophils in five hours after the injection, conditions returning to normal in about twenty-four hours. The possible explanation for this difference is in the toxic effect of the diphtheria bacilli on the parathyroid glands.

ZENTAY, St. Louis.

Chronic Infections

ARTHUS PHENOMENON IN A SYPHILITIC CHILD. I. H. TUMPEER, A. MATHESON and D. C. STRAUS, J. A. M. A. **96**:1373 (April 25) 1931.

A condition resembling the phenomenon noted by Arthus in animals occurred in a patient treated by the authors, following two intramuscular injections of 1,000 units and 15,000 units of diphtheria antitoxin three days apart. Three injections of horse serum toxin-antitoxin had been given four years before, and the first injection of antitoxin during the present illness produced a definite reaction at the time the larger dose was given. The resulting lesions began with swelling, blanching, tenderness and purpuric spots, followed by temporary clearing and later by a purplish-black, depressed, insensitive eschar which soon became necrotic. Death ensued. Extreme care should be exercised in administering serum in the presence of a reaction from a preceding injection of the same type of serum. The syphilitic infection present in this child was probably incidental.

BONAR, Salt Lake City.

THE ULTRAMICROSCOPIC PRECIPITATION REACTION IN SYPHILIS. A. M. MALLOY and R. L. KAHN, J. Infect. Dis. **48**:243 (March) 1931.

The authors note that the more potent a syphilitic serum, the greater is its tendency toward an ultramicroscopic aggregation of antigenic particles, while in

antigen suspension, combined with nonsyphilitic serums, it has a tendency toward dispersion of the antigenic aggregates. Another interesting thing was the fact that shaking the serum and antigen suspension completes the reaction between serum and suspension, tending thus to stabilize the system, limit further change and abolish the need of incubation. The aggregates in the antigenic suspension and the precipitates formed in positively reacting syphilitic serum appear the same ultramicroscopically.

TOOMEY, Cleveland.

PHOSPHORUS, TOTAL CALCIUM AND DIFFUSIBLE CALCIUM CONTENT OF THE BLOOD SERA OF LEPERS AND THEIR RELATION TO BONE CHANGES. J. G. WOOLEY, Pub. Health Rep. **46**:641 (March 20) 1931.

The calcium and inorganic phosphorus content of the blood serum was found to be normal in forty-seven lepers. In fifty-three lepers the diffusible calcium and the percentage of total calcium that was diffused averaged considerably below normal. Roentgenographic studies of the hands and feet showed atrophy of the bone (decalcification or absorption) in 91 per cent.

SANFORD, Chicago.

THE ARNETH FORMULA IN PULMONARY TUBERCULOSIS. DUPONT, Arch. d. mal. du coeur **23**:768 (Dec.) 1930.

The author reviews the technic of making an Arneth count and recalls that in active tuberculosis the formula deviates to the left. When the count is above 200 he regards the prognosis as favorable, when around 170 as poor and below 170 as very poor. This study was carried out on fifty-five patients with pulmonary or pleural tuberculosis. The Arneth counts were made at monthly intervals. The fifty-five cases were divided into four groups. 1. Active progressing tuberculosis with a grave prognosis. This category included twenty-six patients. The highest count was below 170. 2. Cicatricial tuberculosis or stabilized lesions. In this class there were twelve patients. All had counts above 200. 3. Active healing tuberculosis with a good prognosis. There were thirteen patients in this group. All had a shift to the left at first, but the Arneth counts returned to normal as the lesions healed. 4. Fibrocaseous tuberculosis with a good prognosis complicated by relapses. There were four patients in this group.

The author believes the Arneth formula to be so sensitive that it will indicate an occult activity of the tubercle bacillus that no other symptom will reveal. He also believes that the formula reflects the activity of the tuberculous lesions and not the state of resistance of the patient. Under the influence of certain infectious agents (puerperal sepsis, scarlatina, pneumonia, etc.), modifications of the polymorphonuclears occur, but the Arneth formula is not modified by noninfectious maladies (cerebral hemorrhage, asthma, cardiac insufficiency, uterine cancer, Laennec's cirrhosis, etc.). In active advancing tuberculosis with a poor prognosis the presence of infectious disease causes the Arneth count to shift to the right. In healing tuberculosis, infectious processes will cause a shift to the left.

TURNER, Ann Arbor, Mich.

IRRADIATED ERGOSTEROL IN THE TREATMENT FOR TUBERCULOSIS: CLINICAL AND EXPERIMENTAL. F. MEERSSEMAN and C. TRICAULT, Compt. rend. Soc. de biol. **105**:398 (Nov.) 1930.

Irradiated ergosterol appears to produce an increase in the calcification of normal as well as of pathologic tissues, especially in tuberculous lesions. Eleven patients with pleural, peritoneal and glandular tuberculosis received 30 drops of 100 D viosterol (5 mg. of ergosterol) daily in addition to the usual hygienic care. More rapid improvement occurred in their general condition, the milder cases responding more readily than the severe types. Four guinea-pigs were given intraperitoneally 0.5 mg. of Petroff's culture (twenty million tubercle bacilli); two received no viosterol, and died on the fourteenth and twenty-seventh day,

respectively. One animal surviving on the fifteenth day received 3 drops of viosterol daily; its weight increased for a time, but the infection spread, and death ensued on the fortieth day. The fourth animal received the same dose of viosterol, beginning on the same day as the injection of the bacilli; its weight steadily decreased for two months and then increased for twenty-three days, but the infection spread and death occurred after three and one-half months. The pathologic findings consisted of typical peritoneal disease in the first two animals; in the third, more extensive lesions were found in the liver and spleen, while in the fourth guinea-pig the lungs were involved, with a definite sclerosis around all the tuberculous lesions.

WAHL, Cleveland.

THE PRINCIPAL EARLY TYPES OF PULMONARY TUBERCULOSIS IN CHILDREN AND THEIR DIAGNOSIS. P. F. ARMAND-DELILLE and C. LESTOCQUOY, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 468.

There is pathologic evidence of four types of pulmonary tuberculosis in the child: (1) caseous nodules (usually in a lower lobe), (2) inflammation of the lobes of a bronchopneumonic type, (3) caseous lobar pneumonia and (4) ulcerative tuberculosis with cavity. A discussion is given of the symptoms, roentgen findings, course and diagnosis. In conclusion, the author states: "The specific skin tests for tuberculosis are not of themselves a proof that the pulmonary symptoms are tuberculous. The only pathognomonic symptom is the finding of bacilli in the sputum."

HIGGINS, Boston.

MIXED TUBERCULOUS AND MENINGOCOCCIC MENINGITIS. R. C. AGUIRRE and S. I. BETTINOTTI, Arch. argent. de pediat. **1**:611 (Dec.) 1930.

The authors report a case of mixed tuberculous and meningococcic meningitis in a child, 11 months of age, who was brought to the hospital because of a purulent discharge from both ears, coryza and cough, fever and anorexia, rigidity of the neck and a slight opisthotonus of two months' duration. Physical examination revealed a convergent strabismus, muscular twitching and convulsions, moist râles in both lungs, otitis media, splenomegaly and hepatomegaly. The tuberculin reaction was negative. Gram-negative diplococci with characteristics of meningococci were isolated from the pus of the ears and the cerebrospinal fluid; the latter also showed a definite lymphocytosis. The child died ten days later in spite of the treatment with antimeningococcic serum. Autopsy revealed the presence of a generalized miliary tuberculosis with caseo-ulcerative lesions in the apex of the lungs, meninges and mesenteric and tracheobronchial lymph nodes.

According to various statistical records, this type constitutes only 1.4 per cent of all forms of meningitis.

SCHLUTZ, Chicago.

INFANTILE TUBERCULOSIS. EDITORIAL, Bol. d. Cons. nac. de hig., 1930.

Contagion is the cause of infantile tuberculosis and occurs because of tolerance. Tuberculin tests and quarantine of tuberculous persons will do away with these infections. The treatment consists of prophylaxis by hygienic measures and vaccination with B C G.

SANFORD, Chicago.

SPLENECTOMY AS THE ONLY TREATMENT IN BANTI'S SYNDROME DUE TO SYPHILIS. LUIS LEON RESIO, Prensa méd. argent. **17**:656 (Sept. 30) 1930.

Resio reports a case of Banti's syndrome in a girl, aged 17, on whom a splenectomy was performed. Pathologic findings were those of visceral syphilis. The patient received only minor antisyphilitic treatment postoperatively and was apparently normal five years later.

HARRISON, Iowa City.

EXPERIMENTAL CONGENITAL SYPHILIS IN RABBITS. J. A. FINKELSTEIN and S. S. ORLOW, Dermat. Wchnschr. **92**:203 (Feb. 7) 1931.

The offspring of syphilitic rabbits, infected by the cutaneous or intrascrotal method, showed no signs of syphilis and were readily affected by a new infection. Intravenous injections of spirochete emulsions in pregnant females occasionally resulted in intra-uterine infection. Repeated pairing of a healthy male with a syphilitic female sometimes resulted in infection of the male.

TAUSSIG, San Francisco [ARCH. DERMAT. & SYPH.]

CASE OF PRIMARY ACTINOMYCOSIS OF THE LUNGS. L. MOELLER, Monatschr. f. Kinderh. **49**:134 (Feb.) 1931.

The author gives the clinical history and the roentgen and postmortem observations in a boy aged 8 years. There were metastatic abscesses in the liver and brain.

GERSTLEY, Chicago.

THE THROMBOPENIC CURVE IN INDUCED MALARIA. W. LESIGANG and E. ZIPKIS, Monatschr. f. Kinderh. **49**:300 (March) 1931.

In six children with congenital cerebrospinal syphilis, malaria was induced therapeutically. In all cases the thrombocyte count ran a similar course. At the onset of the febrile period it dropped to approximately 30,000 to 50,000. It remained low during the febrile period and returned to normal following the institution of treatment for malaria in about ten days. No explanation is given. The children were from 5 to 14 years of age. An adult, aged 35, was not studied in such detail but showed the same general reaction.

GERSTLEY, Chicago.

THE "FILTRABLE FORM" OF THE CAUSATIVE AGENT OF TUBERCULOSIS. G. W. SCHMIDT, Ztschr. f. Hyg. u. Infektionskr. **112**:95, 1931.

In a lengthy paper, in which a full account of the literature is given, Schmidt reports his negative results in a large series of experimental animals. He was unable to find any evidence of the existence of a pathogenic filtrable or invisible form of the causative agent of tuberculosis (the "ultravirus of tuberculosis"). He believes that the positive results of many workers have been obtained with the help of numerous hypotheses and contradictions. In samples of old tuberculin he found numbers of dead bacilli, which, however, showed no change in form or staining.

FARBER, Boston.

MIXED INFECTIONS OF THE MENINGES; TUBERCLE BACILLI AND OTHER PATHOLOGIC BACTERIA. I. S. PIPIRS, Ztschr. f. Kinderh. **50**:161, 1930.

Mixed infection of the meninges by tubercle bacilli and other pathogenic germs is by no means rare, as it has been assumed to be in Germany. In the majority of cases it is caused by the meningococcus of Weichselbaum, as, for example, in the case reported in this article. The disease seemed to be that of an epidemic meningitis, but numerous tubercle bacilli were found in the spinal fluid, in addition to meningococci. In spite of treatment with antimeningococcal serum the child died, as did all the other patients whose cases were reported in the literature. The disease picture is usually that of a purulent meningitis, which shows, however, the characteristic signs of a mixed infection: (1) the lymphocytosis in the spinal fluid which is found at the height of the disease and (2) the surprising length of time the spinal fluid remains clear. The lymphocytosis represents a general reaction of the tuberculous organism, since it also occurs in tuberculous persons who have a purulent but not a tuberculous meningitis. By a critical observation of the cases described, with regard to the clinical picture, the bacterial

examination of the spinal fluid and the pathologico-anatomic changes, it appears that the tuberculous infection of the meninges is primary in the majority of cases. The reason why so few cases of mixed infection have been found in Germany is probably that the fluid was not searched carefully enough.

EMERSON, Boston.

Internal Diseases

NATIVE INFESTATION WITH DIPHYLLOBOTHRIUM LATUM (FISH TAPEWORM), WITH A REPORT OF FIVE CASES IN CHILDREN. I. PILOT and I. M. LEVIN, Am. J. M. Sc. **181**: 710 (May) 1931.

Five more cases of fish tapeworm are added to twenty-one previously reported. All five patients were Jewish, as were sixteen of the twenty-one. Twenty-one of the twenty-six cases were in children from 3 to 15 years of age. Sampling raw fish while it was being prepared served as the source of infection in three of the five. The life cycle of the broad tapeworm is given as well as the treatment of Osborne and Fishbein, consisting of a liquid diet, a cathartic mixture and oleoresin of *Aspidium*.

HENSKE, Omaha.

OSSIFICATION OF THE COSTAL CARTILAGES: THEIR RELATION TO HABITUS AND DISEASE. FRANK RIEBEL, Am. J. Roentgenol. **21**:44 (Jan.) 1929.

The ossification of the costal cartilages in 541 cases was analyzed and correlated with the incidence of tuberculosis and syphilis and habitus. Ossification proceeds more rapidly in the male. No relationship with tuberculosis, either as an etiologic factor or as a result, was noted. No relation to habitus (hyposthenic, sthenic and hypersthenic) was discovered. Extensive ossification appeared to be more frequent in syphilitic patients, although the wide range in the stages of ossification found at any age makes this sign of limited value in the individual case.

STULIK, Chicago.

LOBAR AND CENTRAL PNEUMONIAS IN CHILDREN. F. B. STEPHENSON, Am. J. Roentgenol. **21**:571 (June) 1929.

To obtain roentgenograms of children's chests of good diagnostic quality requires instantaneous exposure, with regulation of the voltage to the size of the infant or child and making the exposure at a definite phase of respiration, preferably inspiration. The use of high voltage will blot out early shadows of initial inflammatory congestion. While some point to differences in the lobar shadow which may indicate a distinct etiology, the author has been able in the small lung fields to differentiate only between complete lobar, conglomerate lobular and, in some cases, clearing lobar pneumonia. Early stages of lobar pneumonia are so like other inflammations that do not result in clinical lobar pneumonia that one should have in mind always as a basis for diagnosis the physical progressive changes of inflammation of the lungs in general. Formulated rules: (1) Do not predict the outcome of inflammatory shadows or spots. (2) Do not expect classic demarcation of all pneumonias that are clinically lobar or fibrinous pneumonia. (3) Insist on lateral views with the affected side to the plate. (4) Refuse an overexposed plate in which shadows of the congestive stage are blotted out.

STULIK, Chicago.

NIEMANN-PICK'S DISEASE WITH CHERRY-RED SPOTS IN THE MACULA. I. GOLDSTEIN and D. WEXLER, Arch. Ophth. **5**:704 (May) 1931.

The authors report a case of Niemann-Pick's disease in a 10 year old Jewish girl with the typical cherry-red spots in the macula as in amaurotic family idiocy. The child died, and autopsy revealed an enlarged liver, spleen and lymph nodes.

Almost the entire lymphatic structure was replaced by an accumulation of foam cells. In the brain was a marked degeneration of the parenchymatous elements.

WESCOTT, Chicago.

THE THERAPEUTIC USE OF MIXED RESPIRATORY VACCINES. ROY P. FORBES, Arch. Pediat. **48**:48 (Jan.) 1931.

The author questions the value of mixed respiratory vaccines as a prophylactic, but feels that certain favorable results can be obtained when mixed respiratory vaccines are used as a therapeutic agent.

This method of treatment was used in the following types of respiratory infection: acute and subacute rhinopharyngitis, influenza with relapse and cough, pneumonia, subacute bronchitis and asthmatic bronchitis.

The author states that no lasting immunity is conferred by this form of therapy. He is of the opinion that the apparent efficiency of vaccine therapy may be the effect of nonspecific protein.

WADDELL, University, Va.

EXOPHTHALMIC GOITER IN A GIRL THREE AND ONE-HALF YEARS OF AGE. CHARLES GILMORE KERLEY, Arch. Pediat. **48**:61 (Jan.) 1931.

The author gives the physical observations in a case of exophthalmic goiter in a girl, aged 3½. Treatment consisted of rest and the administration of potassium arsenite, a solution of peptonate of iron and tincture of iodine compound. Rapid improvement followed six months of rest.

WADDELL, University, Va.

A CASE OF PRIMARY SARCOMA OF THE LUNG IN AN INFANT TWENTY-NINE MONTHS OF AGE. PHILIP ROSENBLUM and BENJAMIN GASUL, Arch. Pediat. **48**:63 (Jan.) 1931.

The authors found only two cases of primary sarcoma in infancy reported in the literature. They present the history, physical indications and roentgenologic and pathologic observations in a case occurring in an infant, 29 months of age.

WADDELL, University, Va.

THE USE OF RESPIRATORY VACCINE IN THE TREATMENT OF PNEUMONIA IN CHILDREN. ROY P. FORBES and CHARLES L. STEINBERG, Arch. Pediat. **48**: 238 (April) 1931.

The authors report fifty cases of pneumonia in children treated with large doses of mixed respiratory vaccine administered at six hour intervals. As a control group, sixty-six cases not so treated are reported. In the children treated with vaccine a mortality of 18.7 per cent was noted, as compared with a mortality of 27.3 per cent in the control series. When treatment was instituted within forty-eight hours of the onset the mortality was 4.6 per cent as compared with 20.6 per cent in the control series. The authors feel that the treatment possesses real value especially in early pneumonia. The results obtained by these workers are very similar to those obtained by Lambert and by Sutton in adults.

WADDELL, University, Va.

JUGULAR BULB THROMBOSIS: A CASE WITH RECOVERY FOLLOWING LIGATION OF THE INTERNAL JUGULAR VEIN, METAPHEN AND ANTISTREPTOCOCCIC SERUM. GEORGE J. FELDSTEIN and H. A. SLESINGER, Arch. Pediat. **48**: 259 (April) 1931.

A case of thrombosis of the jugular bulb is presented. Recovery followed ligation of the internal jugular vein and the administration of metaphen and antistreptococcic serum. In addition to the case report there is a discussion of the condition from the standpoint of incidence, etiology, pathology, symptoms, diagnosis and treatment.

WADDELL, University, Va.

A Further Note on "Status Thymico-Lymphaticus." M. Greenwood, J. Hygiene **30**:403 (Nov.) 1930.

Professor Hammar's careful and detailed work is quoted and analyzed in support of the author's belief that heavy thymi are not the cause of sudden inexplicable deaths. Such deaths occur no more frequently in persons with heavy thymus glands than in others. In a group of children dying from unexplained causes the thymus was studied as to weight, Hassall bodies and ratio of cortex to medulla. Histologic abnormalities demonstrable in individual cases that might be of diagnostic aid did not exist. "So far, then, as practical affairs are concerned, we may be quite confident that Status-Thymico-Lymphaticus, etc., is a mere legal fiction."

Janney, Milwaukee.

The Electrocardiogram in the Healthy Child. G. Halfer, Riv. di clin. pediat. **28**:959 (Nov.) 1930.

The author gives reports of electrocardiographic studies of ten well children between 15 months and 9 years of age. The following deductions are drawn:

The electrocardiogram of the child is distinctive. There is an accentuation of all the positive waves. After the third year of life the P-R interval becomes normal. The R wave increases progressively to reach that height observed in adults.

The S wave is a little deep. The T wave is variable. In half of the patients and especially in those under 3 years of age there was a right ventricular preponderance; in others there was equilibrium between the two ventricles.

Higgins, Boston.

Chronic Infantile Polyarthritis and Still's Disease. A. Gentili, Riv. di clin. pediat. **28**:1017 (Nov.) 1930.

The author reports four cases of Still's disease. Still's disease is rare in Italy. It is a form of chronic polyarthritis of children always accompanied by enlargement of the spleen and hypertrophy of the lymph glands, and frequently by enlargement of the liver; in the author's opinion, the foregoing characteristics justify its being differentiated from the common type of infantile polyarthritis.

Higgins, Boston.

Purpura Hemorrhagica from Streptococci. E. de Micheli, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 227.

In a boy, aged 4 years, a fulminating "purpura haemorrhagica" developed, twenty days after an attack of scarlet fever. A hemorrhagic nephritis with anuria was present. There was bleeding from the mouth and nose and many ecchymoses and petechiae were present in the skin. The child died two days after the appearance of the hemorrhagic symptoms. A necropsy was performed. On culture of the heart's blood, a gram-negative coccus — mostly in pairs, but occasionally in chains — was obtained. Rabbits inoculated with the culture died of a hemorrhagic syndrome; the lesions were similar to those in the patient.

[Abstractor's Note: Meningococcus septicemia (spotted fever) should be considered in the differential diagnosis of this case.]

Higgins, Boston.

The Treatment for Purulent Pleurisy by Ethylhydrocuprein. P. Woringer, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 456.

The author has followed essentially the procedure outlined by Gralka in treating patients for pneumococcus empyema with ethylhydrocuprein. His procedure is: "One withdraws by thoracentesis as much pus as possible and then injects

25 mg. of optochin chloride (0.5 cc. of a 5 per cent solution) per kilogram of body weight, but never more than 0.500 Gm. (10 cc.) at one time. One can, previously to this injection, irrigate the pleura with a 0.5 per cent solution of ethylhydrocuprein. The treatment is repeated three or four times at one or two day intervals and then stopped. Often the temperature then becomes normal and the condition of the child shows improvement; in this case, one quietly awaits the spontaneous absorption of the rest of the fluid. If, on the other hand, the fever returns, one uses a second series of injections of ethylhydrocuprein and even a third and a fourth, if occasion demands." Of thirty-six children treated by this method, twenty-four were completely cured without other treatment; five required a secondary operation. In one a secondary staphylococcus empyema developed, and six died. Thus the percentage of deaths was 16.7; 67 per cent were cured by ethylhydrocuprein therapy alone. In a comparison of his results with the data given by three other writers who used surgical therapy, the author gives the following percentages of fatal cases:

Age	Ethylhydro-cuprein	Operation		
		I	II	III
Under 1 year	28	88	64	41
1–2 years	36	62	57	43
2–3 years	0	86	14	22

He makes the following comments on this method of treatment: The action of ethylhydrocuprein is limited to the cavity in which it is injected; in practice, it never has any effect on foci at a distance. In practice, one cannot expect a cure by this treatment unless the pneumococcus is the only cause of the empyema. When, after a first series of injections, the fever returns and pneumococci reappear in the fluid, it is not too late for treatment with ethylhydrocuprein. It is impossible to predict the number of injections that will be necessary. Some patients are resistant to the drug, but fortunately these cases are rare. Even when surgical treatment becomes necessary, the injection has always been of some help. In no case was any ocular trouble, such as optic neuritis or blindness, observed which could be traced to ethylhydrocuprein.

HIGGINS, Boston.

AEROPHAGIA IN THE CHILD. G. GUIDI, Scritti med. dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 567.

Aerophagia may be due to the swallowing of air with the saliva, usually in excessive amounts, or to the mechanism of contracting the diaphragm suddenly with the glottis closed. The author reports a case in a boy, aged 12 years, who suffered from loss of appetite, vomiting, distention, colic and abdominal distress due to swallowing air. The condition was apparently caused by excessive salivation, due in turn to wires recently applied to straighten the teeth. Treatment consisted of the explanation of the symptoms to the boy and his parents, and his attempt to avoid swallowing air. A noteworthy improvement took place in a few days.

HIGGINS, Boston.

AN ELECTROCARDIOGRAPHIC OBSERVATION IN AN INFANT DYING AFTER THE INJECTION OF MUSCARINE. F. HURTADO and J. C. CALDERIN, Bol. Soc. cubana de pediat. **3**:59 (Feb.) 1931.

An electrocardiogram taken in an infant, aged 9 months, in a preagonal state from a severe gastro-intestinal intoxication, showed: A resistance of less than 2,000 ohms, a left ventricular preponderance and a sinus tachycardia of 200 beats per minute. The following alterations were observed after an intramuscular injection of 4 cc. of muscarine: The resistance equaled 0. The cardiac rate was 175 per minute. In lead I, the P wave was irregular or absent. The ventricular

complex was bizarre and resembled a partial ventricular block or a right and left bundle branch block. In lead II, the P wave was present in all complexes. The St space originated above the iso-electric line and descended toward it, forming a marked upward convexity. The ventricular complex was the same as that in lead I. In lead III, the P wave was similar to that in lead I. The ventricular complex presented a partial alternating block of the right and left bundle branches. The authors arrive at no conclusions as to the nature of these alterations in the electrocardiogram.

SCHLUTZ, Chicago.

THE DIAGNOSIS AND TREATMENT OF GOITER IN NURSLINGS. K. RUPILIUS, Arch. f. Kinderh. **91**:173 (Sept. 19) 1930.

Twenty-eight cases of goiter in children under 18 months of age have been studied by the author. The clinical symptoms are dyspnea, catarrh, dysphagia and stridor. Enlargement is grossly observable in only one half of the cases. In the remainder the condition is usually retrosternal and can be diagnosed by percussion, roentgenograms and intensification of the dyspnea when the head is extended. The treatment employed by the author consisted of small doses of iodides (0.01, 0.1 and 1.0 mg.). The symptoms returned in 20 per cent of the cases, but disappeared under another course of treatment. Frequently one dose of iodides stimulates the metabolism so that the goiter disappears.

SANFORD, Chicago.

STUDIES ON SERUM-SICKNESS. W. BALABAN, Arch. f. Kinderh. **91**:193 (Sept. 19) 1930.

A study was made of 33 cases of serum sickness developing during the year 1928 following injections of antimeningococcus serum. Repeated injections of serum increase the allergic condition. The first attack in a susceptible child is determined by the constitutional condition, but subsequent attacks depend on the length of time of injections. The nervous system, blood, blood vessels and glandular organs are involved. The changes in the nervous system are in the vasomotor apparatus; those in the blood occur as leukopenia and hemorrhagic manifestations. For the underlying pathologic condition for which the serum is given, the reaction to the anaphylactic shock is either negative or favorable.

SANFORD, Chicago.

A CASE OF MALIGNANT RENAL TUMOR IN INFANCY. H. STUX, Arch. f. Kinderh. **92**:128 (Dec. 19) 1930.

A boy, 10 months old, presented symptoms of a mass in the abdomen. Operation revealed a large renal tumor. There were no signs except a microscopic hematuria.

SANFORD, Chicago.

ATYPICAL COURSE IN A CASE OF LYMPHOGRANULOMATOSIS. WOLFGANG STREMPEL, Inaug. Dissert., Kiel, 1929.

An illness in a boy, aged 3½, observed for four months, was characterized by severe anemia without signs of regeneration, leukopenia with almost complete disappearance of the granular elements, relative and absolute lymphocytosis, thrombopenia, hemorrhagic diathesis, an enlarged liver, a large smooth spleen and septic phenomena with fever. Marked enlargement of the lymph nodes appeared only a few days before death. Necropsy showed marked proliferation of reticuloendothelial cells, lymphocytes and giant cells in the lymph glands, spleen, liver, bone marrow, kidneys and testicles. The finding of many cocci in the testicles is interpreted as probably due to infection secondary to the leukopenia. The differential diagnosis and other cases from the literature are discussed.

CRANE, Chicago.

Nervous Diseases

CEREBRAL BIRTH CONDITIONS, WITH SPECIAL REFERENCE TO CEREBRAL DIPLEGIA. CLARENCE A. PATTEN, Arch. Neurol. & Psychiat. **25**:453 (March) 1931.

Forty-six cases of cerebral diplegia were analyzed to determine whether they should be considered as developmental defects, disorders of growth or due to maternal diseases, hereditary influences or traumatization of the nervous system. Patten points out that a rather large percentage of these children have irrelevant birth history and that there is no specific reason for considering the condition due to birth injury. He points out that interference with myelinization might come about through alteration of the blood from toxic or infectious conditions in the mother, and in postnatal life through improper food. These conditions may give rise to motor involvement, which clinically cannot be distinguished from that due to birth injury.

BEVERLY, Chicago.

DIFFUSE PROGRESSIVE DEGENERATION OF THE GRAY MATTER OF THE CEREBRUM. BERNARD J. ALPERS, Arch. Neurol. & Psychiat. **25**:469 (March) 1931.

This is a detailed microscopic study of the pathologic process in an infant 3 months of age. The cause of death was birth injury with subnormal mentality. The anatomic substratum consisted of a marked demyelinization of the cortex, due probably to a failure of development, and a similar, though less marked, absence of fibers in the basal ganglions. The cortex showed a diffuse loss of ganglion cells in the third lamina. These observations are compared with those of other authors.

BEVERLY, Chicago.

THE BRAIN AND THE CEREBROSPINAL FLUID IN ACUTE ASEPTIC CEREBRAL EMBOLISM. WILLIAM CONE and S. E. BARRERA, Arch. Neurol. & Psychiat. **25**:523 (March) 1931.

The authors studied the pathologic picture of aseptic cerebral embolism in dogs and three human subjects. Polymorphonuclear leukocytes pour out into the infarcted area of the brain and into the overlying meninges eight and a half hours after the vessels of that part are blocked. They increase in number up to forty-eight hours. At seventy-two hours the cells show progressive evidences of degeneration, and at the end of seven days they have been entirely replaced by compound granular corpuscles. During the early phases the leukocytes may reach the cerebrospinal fluid in varying numbers by way of the perivascular spaces, by direct invasion of the subarachnoid space from an adjoining area of softening or, when the infarcted area borders the ventricle, by extension directly into the ventricular fluid. The authors suggest that possibly some of the cases recorded in the literature as acute hemorrhagic encephalitis may have been of embolic origin.

BEVERLY, Chicago.

INTRAMEDULLARY TUMORS OF THE SPINAL CORD. JAMES W. KERNOHAN, HENRY W. WOLTMAN and ALFRED W. ADSON, Arch. Neurol. & Psychiat. **25**:679 (April) 1931.

This is a detailed histologic report of fifty-one cases of intramedullary tumors of the spinal cord. Tumors are classified according to the predominating type of cells present. The average duration of the symptoms was 4.9 years. A dissociated waistcoat type of sensory disturbance and a marked difference in the levels of various sensory disturbances suggests intramedullary tumor. Involvement of the lower motor neurons was present in 40 per cent of the cases; root pains referred to a distance from the cord, present in 48 per cent, were less common than when extramedullary tumors were present. Spinal pains, present in 72 per

cent, were much more common than in cases of extramedullary growths. In only a few cases could the tumors be removed completely.

BEVERLY, Chicago.

THE CEREBELLUM. AUBREY T. MUSSEN, Arch. Neurol. & Psychiat. **25**:702 (April) 1931.

The function of the three lobes of the cerebellum was studied in cats by electrical stimulation and destruction of the lobes. The author concluded that the anterior lobe is associated with forward balance, the posterior lobe regulates backward balance and the middle lobe is concerned with the rotation movements of the head. While the cerebellum acts as a whole, the primary reflex depends on the lobe stimulated.

BEVERLY, Chicago.

PINEALOMAS. JOSEPH H. GLOBUS and SAMUEL SILBERT, Arch. Neurol. & Psychiat. **25**:937 (May) 1931.

The authors studied the development of the pineal gland from the fifth prenatal month to the fifth year of life. During intra-uterine life the body has glandular characteristics which become particularly striking at about the fourth month of postnatal life. In later stages of differentiation it becomes very vascular. During the early months of life it loses its glandular structure.

Seven cases of pinealomas are reported. All cases presented the usual symptoms of tumor. In only one case was any unusual genital development noted. Five of the patients gave a history of diplopia. Argyll Robertson pupils (complete or incomplete) were present in six cases and in all of these were accompanied by a paresis of the muscles of the eyes. Interference with conjugate movement of the eyes, particularly limitation of upward gaze, is the most important localizing symptom in tumors of this region. Bilateral symmetrical hydrocephalus was found in every case.

After a careful histologic study of these cases the authors conclude: "We can with certainty trace the tumors, dissimilar as they appear to be in structure, to the same origin. In the microscopic make-up of the pineal gland in the third or fourth month of postnatal life, we find the histologic nucleus about which the variable histologic pictures of any tumor of the pineal type can be grouped."

BEVERLY, Chicago.

ENCEPHALITIC, IDIOPATHIC AND ARTERIOSCLEROTIC PARKINSONISM. MOSES KESCHNER and PAUL SLOANE, Arch. Neurol. & Psychiat. **25**:1011 (May) 1931.

In this careful histologic study of parkinsonism the changes in the basal ganglions did not differ. There is not a close correlation between the clinical data and course of the disease and the histologic observations.

BEVERLY, Chicago.

ENCEPHALOPATHIA PERIAXIALIS DIFFUSA (SCHILDER'S DISEASE). CHARLES DAVISON and WILLIAM SCHICK, Arch. Neurol. & Psychiat. **25**:1063 (May) 1931.

The clinical and pathologic picture of an unusual case of Schilder's disease in a 15 year old girl is reported. The case was unusual in having an intense optic atrophy and extrapyramidal disorders with lesions in the nuclear masses and fiber tracts of the putamen and globus pallidus.

BEVERLY, Chicago.

EPILEPSY OF THE ANGIOSPASTIC VARIETY IN MONOZYGOTIC TWINS. D. M. OLKON, Arch. Neurol. & Psychiat. **25**:1111 (May) 1931.

Genuine epilepsy was reported in identical twins. Studies of the capillaries revealed extensive hemorrhages in the superficial capillaries following convulsions

indicating a violent angiospasm. A diagnosis of spasmophilic epilepsy was therefore made. Antispasmodic medication (tincture of belladonna and calcium lactate) greatly reduced the attacks, and both boys finally went three years without an attack. This treatment was tried after phenobarbital, bromides and a ketogenic diet had failed to control the convulsions.

BEVERLY, Chicago.

ACUTE DIFFUSE ENCEPHALITIS. GEORGE B. HASSIN and SAMUEL J. HOFFMAN, Arch. Neurol. & Psychiat. **25**:1118 (May) 1931.

The authors report two cases of acute diffuse encephalitis with partial recovery. In both cases a marked papilledema of the optic nerves developed, suggesting tumor of the brain. In one case the condition subsided entirely; in the other, a partial optic atrophy remained.

BEVERLY, Chicago.

THE APPROACH TO COLLEGE MENTAL HYGIENE. K. E. APPEL and L. H. SMITH, Ment. Hyg. **15**:52 (Jan.) 1931.

Although many of the claims made for mental hygiene seem excessive, there is available a modicum of definite knowledge, which Appel and Smith think should be included in the curriculum of every college student. In the absence of such information the individual is often vexed with the dilemmas of his personality and handicapped by the results of his emotional strife. And yet, in the seven cases reviewed by Appel and Smith, six of the patients turned away from psychology because it touched on sensitive spots, and to one the untutored effort to gain knowledge was detrimental rather then helpful.

The suggested program of mental hygiene consists of: carefully selected, introductory reading both in literature and in psychology; a comprehensive series of lectures on psychoanalysis, behaviorism, Gestalt psychology, endocrine physiology and psychoneuroses, and the correlation of thorough social histories of the students with their health and scholastic records. A committee, composed of the dean and the interested members of the faculty and assisted by a psychiatrist, could cope with this task. Tactful work by this committee should prove of considerable help in alleviating the maladjustment, so common in college life, and might perhaps prove of value in preventing emotional conflicts in the future.

DAVIDSON, Newark, N. J. [ARCH. NEUROL. & PSYCHIAT.]

A STUDY OF SOME SCHIZOID CHILDREN. A. T. CHILDERS, Ment. Hyg. **15**:106 (Jan.) 1931.

A striking similarity of certain manifestations among a group of problem children to the early symptoms of adult psychoses, and particularly to incipient schizophrenia, prompts Childers to term this type of child "schizoid." Of 114 children under observation, 19 belong to this type. A "schizoid" child lives in a world of exaggerated fantasy. Whereas the life of fantasy, however vivid, of most children disappears with change of mood, with the "schizoid" child it assumes the importance of reality and often develops into mental trends. Hallucinations are common among the "schizoid" group, and there is a sinister definiteness about them. These children desire to be of the opposite sex or to retrogress in age; their dreams are crowded with ideas of escape and of bizarre bodily changes. Childers observed that most of the "schizoid" children were reared by foster parents, or had an unusual position in the family. There seems little doubt that in adulthood these children will manifest schizophrenia.

DAVIDSON, Newark, N. J. [ARCH. NEUROL. & PSYCHIAT.]

THE EARLY DIAGNOSIS AND SERUM TREATMENT OF POLIOMYELITIS. K. T. HELMS, M. J. Australia **1**:71 (Jan. 17) 1931.

The histopathology of experimental poliomyelitis as described by Fairbrother and Hurst is reviewed. Three types of diseases are recognized: the abortive,

the nonparalytic and the paralytic. The most typical early symptoms are given as fever, one or two attacks of vomiting, drowsiness, irritability when roused and retention of urine. The characteristic signs are the spine sign (unwillingness to flex the spine) coarse tremors, hyperesthesia and an asymmetrical reflex disturbance. The difficulties of early diagnosis would seem to be emphasized by the report of 143 cases in which the condition was suspected. In none of them did paralysis develop, and in most cases another diagnosis was established.

The author advised against spinal puncture unless immune serum can be given at once, as it has been shown that invasion of the central nervous system will follow the intravenous injection of virus if the subarachnoid space is damaged.

The use of convalescent serum is based on conclusive experimental work which demonstrated that a virulent culture could be made harmless by mixture with serum and that an attack could be prevented by the intravenous injection of serum as late as twenty-four hours after the intracerebral inoculation of virus. The same results could not be obtained with Rosenow's serum. Favorable results in the clinical use of convalescent serum in Massachusetts and in Australia are given.

TENNEY, Madison, Wis.

SCHILDER'S PERIAXIAL ENCEPHALITIS. C. WORSTER-DROUGHT and T. R. HILL, Proc. Roy. Soc. Med. **24**:320 (Jan.) 1931.

A case of Schilder's periaxial encephalitis is reported in a child 7 years old.

WILLIAMSON, New Orleans.

PROGRESSIVE MUSCULAR ATROPHY OF SPINAL ORIGIN. W. G. WYLLIE, Proc. Roy. Soc. Med. **24**:323 (Jan.) 1931.

A case of progressive muscular atrophy of spinal origin is reported in a child of 2½ years.

WILLIAMSON, New Orleans.

THE CEREBROSPINAL FLUID IN THE DISEASE OF HEINE-MEDIN. G. TACCONE, Scritti medici dedicati a Carlo Comba, ediz., Riv. di clin. pediat., 1929, p. 334.

The author has studied the spinal fluid obtained in forty-seven cases of infantile paralysis at various stages from the first day of the disease to a year and a half later. The results are given in a table. In conclusion he says: "It does not seem possible to establish an exact formula for the findings on the fluid in this disease since it may present characteristics common to the meningeal reactions in different conditions." The cell count and presence of globulin in the early stages of the disease alone are characteristic. In many cases, examination of the spinal fluid is necessary to differentiate poliomyelitis from meningitis of some other type.

HIGGINS, Boston.

MENTAL HYGIENE IN PEDIATRICS. MASSILLON SABOIA, Arch. de pediat., Rio de Janeiro **27**:48 (Dec.) 1930.

Of the objectives of preventive pediatrics as defined by Dr. Border Veeder, Dr. Saboia emphasizes that of the formation of correct physical and mental habits in infancy and early childhood and the need of the early diagnosis of abnormal ones if the formation of neuroses and psychoses is to be avoided in later life. Very early diagnosis is also important in cases of subnormal intelligence due to hypothyroidism, for here early treatment gives the most hopeful results in the normal development of the nervous system. He commends the progress of mental hygiene in pediatrics in North America and in the more advanced countries of Europe, and describes the preventive and corrective work of the child guidance clinics and the habit clinics of Philadelphia, New York, Boston and Minneapolis.

He points out, by way of contrast, the lack of facilities for proper physical and mental development among the children in the larger cities of Brazil, stresses the need of public or private gymnasiums and playgrounds, and proposes the consideration of plans for the establishment of a central institution in Rio de Janeiro having for its purpose both prophylactic pediatrics and child neuropsychiatry.

OLIVER, Columbus, Ohio.

Diseases of the Eye

A CASE OF MICROPHTHALMOS. P. SANDER, Brit. J. Ophth. **15**:22 (Jan.) 1931.

The family history makes this case of bilateral microphthalmos of interest. In the second month of pregnancy the mother watched an operation for acute glaucoma on her mother's eye. The operation was complicated by the copious loss of vitreous and subsequent loss of the eye. It then became a fixed idea with the mother that her baby would have something wrong with its eyes. During pregnancy she frequently experienced apprehension for the eyes of her future child. When born, the infant "had no eyes." The child died of an acute infection.

ZENTMAYER, Philadelphia [ARCH. OPHTH.]

A FAMILY AFFECTED WITH KERATOCONUS AND ANTERIOR POLAR CATARACT. P. SANDER, Brit. J. Ophth. **15**:23 (Jan.) 1931.

In each eye of a man and his three daughters there was an anterior polar cataract in combination with typical keratoconus. The degree of the development of the symptoms increased with the age of the persons examined. All the members of the family presented features of physical and mental degeneration.

The author says that it would be stretching the imagination to apply the hypothesis of infection of the eyes during intra-uterine life with perforation of the cornea and penetration by either microbes, probably gonococci, or their toxins to the lens. It would be more natural to consider the polar cataract as one of the defects of development and nutrition, as is the case in other stigmas of degeneration for which no infection is considered necessary.

ZENTMAYER, Philadelphia [ARCH. OPHTH.]

TWO CASES OF QUININE AMBLYOPIA WITH UNUSUAL OPHTHALMOSCOPIC PICTURE. J. N. DUGGAN and B. P. NANAVATI, Brit. J. Ophth. **15**:160 (March) 1931.

The first case was that of a girl, aged 9 years. For an acute attack of malaria she had taken four doses of 1½ grains (0.09 Gm.) of quinine at four hour intervals the first day and two doses the following day, when she suddenly became blind in both eyes. There were no other symptoms of quinine poisoning. Twenty hours after the onset, the eyes were still totally blind. In both eyes the optic papilla and retinal vessels were normal. There was edema of the retina extending to the periphery of the fundus which looked yellowish white. A faint red spot marked the macula. Four days later the papillae were white, the retinal vessels were much contracted, the fundus reflex was normal, and both macular regions showed glistening yellowish-white dots arranged irregularly around the macula. One week later there was perception of light. The retinal vessels were more contracted, but the macular changes were less marked. Seventeen days later, vision was 6/6. Each field was concentrically contracted to within 10 degrees of fixation. The papillae remained white, and the vessels were contracted, with white lines along them.

The second case was that of a man 20 years of age. He had taken 378 grains (24.49 Gm.) in 2 grain (0.13 Gm.) doses over a period of three weeks. The onset of blindness was sudden. The fundus picture, except for the absence of the macular glistening spots, was the same as in the first case. In seventeen days vision had returned to normal.

Since malaria is so common in the tropics and quinine is so freely used, the author is of the opinion that some of the cases of optic atrophy in which no definite cause can be found may be due to the toxicity of quinine.

ZENTMAYER, Philadelphia [ARCH. OPHTH.]

TREPHINING IN THE TREATMENT OF CONGENITAL GLAUCOMA. R. E. WRIGHT and K. KOMAN NAYAR, Brit. J. Ophth. **15**:166 (March) 1931.

The authors state that in common with other observers they have sometimes found the treatment for congenital glaucoma very disappointing, and it was therefore with some satisfaction that they recently observed the effects of successful trephining in two cases. Posterior sclerotomy as an operative treatment in any condition of the eye has always seemed to them a remedy to be avoided if some less drastic procedure can be adopted. They also reported a somewhat unusual group of cases of congenital glaucoma suggesting familial sex-linked characters.

The success or failure of an anterior decompression must largely depend on whether the normal relationship of the aqueous and vitreous is preserved; the changes that result in the zonule and the vitreous body itself, as a result of stretching of the globe, eventually upset this relationship in certain cases.

ZENTMAYER, Philadelphia [ARCH. OPHTH.]

THE FREQUENCY OF DEFICIENCY DISEASES OF THE EYE DUE TO LACK OF VITAMIN A IN A MILITARY CAMP NORTH OF PEIPING. ARNOLD PILLAT, Nat. M. J., China **15**:585 (Oct.) 1929.

Pillat describes the main ocular symptoms in cases of vitamin deficiency: night-blindness, dryness of the eyeball conjunctiva, loss of luster of the cornea with reduced sensibility, keratomalacia, pigmentation of the conjunctiva, meibomitis, blepharitis, decrease of tear fluid, edema and puffiness of the lids and comedones. All these symptoms disappear with proper nourishment and treatment with cod liver oil.

LUCAS, San Francisco.

OCULAR FINDINGS IN SOME INFECTIOUS DISEASES (TYPHUS, TYPHOID, DYSENTERY, KALA AZAR AND PELLAGRA). W. P. LING, Nat. M. J., China **15**:632 (Oct.) 1929.

In a fifty-page monograph, Ling presents a detailed description of the ocular findings in typhus, typhoid, dysentery, kala-azar and pellagra. An extensive review of the literature in that field is presented.

LUCAS, San Francisco.

PERSISTENCE OF THE HYALOID ARTERY AND THE CANAL OF CLOQUET. DEJEAN, Arch. d'opht. **47**:459 (July) 1930.

Dejean first describes a case in which a persistent hyaloid artery associated with a posterior polar cataract was carefully studied with the slit-lamp. The canal of Cloquet was traceable from the posterior surface of the lens to the disk. He discusses the formation of the vitreous and suggests that Cloquet's canal normally persists in the form of the optically empty retrolental space. He agrees with Vogt in thinking that the arcuate line seen on the posterior surface of the lens is the remnant of an attachment of Cloquet's canal. He reiterates his belief that the vitreous is composed of sheets which contain fluid and therefore should not be cut away when it escapes from a wound.

MARLOW, Syracuse, N. Y. [ARCH. OPHTH.]

CONGENITAL ENTROPION. GOMEZ MARQUEZ, Arch. de oftal. hisp. am. **30**:187 (April) 1930.

After referring to the infrequency of congenital entropion, the author reports two cases. One case was that of bilateral entropion of both lower lids, in which

cure was obtained by excision of a horizontal fold of skin and muscle parallel to the margin of the lid, supplemented in one eye by section of the tarsus, with a good final result. The second case was one of monocular entropion of the lower lid, the margin of which was fixed to the orbital rim. In this case the author made a long incision between the palpebral and the bulbar conjunctiva down to the orbital margin, liberating the lid; a fibrous band which fixed the eyeball to the lower orbital wall was sectioned, and a mucous flap from the mouth was inserted into the breach and fixed with sutures. A slight entropion which persisted was later corrected by transplantation of the internal half of the margin of the lid according to the technic of Spencer Watson, by enlargement of the lower culdesac with transplantation of a mucous flap from the upper lid. Later, after several operations for a coexisting strabismus, a satisfactory result was obtained.

In commenting on these cases, the author differentiates two classes of congenital entropion: (*a*) essential entropion with normal lids, and (*b*) symptomatic entropion which coexists with other palpebral malformations. The first is similar to senile entropion, and the second to cicatricial entropion. The treatment is surgical: either the author's procedure or Aubineau's flap with a pedicle after liberation of the margin lid.

FINLAY, Havana, Cuba.

EXPERIMENTAL STUDIES ON XEROPHTHALMIA. M. RUTHARDT, Arch. f. Kinderh. **90**:252 (June 27) 1930.

The author studied the rôle of vitamins A, B and C in xerophthalmia, as well as the relation of salts to this condition. Vitamins B and C were furnished by yeast and lime juice. White rats were used, the eyes being disinfected daily with boric acid. He found that both vitamins B and C and the salt content were factors; for example, in a vitamin A-free diet, if the salt content was kept low, healing would occur almost as fast as when milk and cod liver oil were added to the diet. The amount of vitamins B and C also influenced the healing.

SANFORD, Chicago.

INTERFERENCE THERAPY (VACCINATION) IN METASTATIC DISEASES OF THE EYE. N. BLATT, Cluj. med. **11**:181 (April 1) 1930.

It is a well known fact that there are cases in which a severe disease assumes a favorable course owing to the intercurrence of a new infectious disease. An explanation of this fact may be derived from the notion of interference; each type of disease, especially its course, is an example of the principle of interference. The fate of the diseased system is a function of the interaction of the waves of vitality. If they annihilate one another in the sense of the interference phenomena, it may result in cure. In general practice one must consider the doctrine of interference. In some instances malaria is cured after an attack of erysipelas, syphilis after influenza, epilepsy after scarlet fever, etc. Within the compass of interference therapy, the experiences gathered at the sickbed in regard to the mutual favorable influence of two coexistent diseases of the system on one another should also be applied to rational therapeutic activity and utilized in practice.

The stimulation therapy of the already routine tuberculin-vaccine treatment also falls within the compass of interference therapy. Malaria therapy, too, acts in the sense of interference therapy. A second method of the interference therapy is vaccination against smallpox. The therapeutic effect of vaccination can be explained in the sense of such therapy, in that the causative agent of smallpox, the resistant virus, is transferred by inoculation. So by vaccination against smallpox one may apply a method by which the general practitioner can also carry out interference therapy. In metastatic purulent diseases of the eye a favorable effect may be expected from such therapy.

In severe cases of metastatic processes of the eye, especially purulent keratitis, iridochoroiditis and metastatic iritis, the patients were treated by vaccination against smallpox. In three cases the healing was complete, and in two, interference therapy proved a failure.

BLATT, Tagu-Mures, Rumania.

Diseases of the Ear, Nose and Throat

THE PHYSIOLOGY OF HEARING. ARNO B. LUCKHARDT, Arch. Otolaryng. **12**:413 (Oct.) 1930.

The author gives a brief discussion of the more important phases of hearing. He compares audition in the various forms of biologic life, with special reference to man and the higher mammals. This is followed by a description of the sound-receiving, sound-conducting and sound-perceiving apparatus of man and a discussion of the more important theories of hearing.

BARNETT, Chicago.

CLEFT PALATE SPEECH. JOHN A. GLASSBURG, Arch. Otolaryng. **12**:820 (Dec.) 1930.

The author's statements are so concise that they are worth quoting: "Cleft palate speech is characterized by a pronounced nasality of all the vowel sounds, an inability to pronounce the dentals *t* and *d*, the labials *p* and *b*, the palatals *k*, hard *g* and hard *c* and an incorrect pronunciations of the labiodentals *f*, *v*, *l*, *r*, and the sibilants *s* and *z*.

"There are several types of cleft palate: a cleft of the soft palate, a cleft of the hard palate, a cleft in both or a cleft in the upper lip. This last condition is known as harelip and is frequently associated with other types of cleft palate.

"The speech defect is due to a failure of function on the part of the palate. The soft palate and its prolongation, the uvula, serve to close up the posterior passage to the nose during phonation. This closure takes place in the production of all the forty-three sound units in the English language, except three, the nasals *n*, *m* and *ng*.

"When there is a cleft of the palate, there is a failure in closure, and the breath stream, instead of being directed downward to issue through the mouth, escapes into the nasal cavities and the speech produced is nasalized or of a snoring quality.

"The treatment of the cleft is surgical and mechanical. The operation is to gap over the palatal space. The earlier the operation is performed, the better the results. When the cleft cannot be surgically closed, dental plates are indicated. But in all cases, speech education is necessary to overcome the defective phonation.

"The principles underlying the educational treatment can be considered under four headings: (1) oral expiration, (2) palatal stimulation, (3) vocal gymnastics and (4) phonetic education. The oral expiration consists of efforts to direct the breath stream out of the mouth instead of out through the nose. Palatal stimulation may be acquired by physical exercises of the palatal muscles, the faradic current and the palatomasseur. Vocal gymnastics are exercises to overcome the spasticity of the lips. Phonetic education is employed to correct the errors of pronunciation."

These methods of treatment are then elaborated on.

BARNETT, Chicago.

MALDEVELOPMENTS OF THE AURICLE, EXTERNAL ACOUSTIC MEATUS AND MIDDLE EAR: MICROTIA AND CONGENITAL MEATAL ATRESIA. J. S. FRASER, Arch. Otolaryng. **13**:1 (Jan.) 1931.

The author's summary is as follows: "1. The external and middle ears are developed from the first visceral or pharyngeal cleft and the first and second visceral arches. The inner or medial part of the first cleft forms the eustachian tube and tympanum, while the outer or lateral part is converted into the auricle and external acoustic meatus.

"2. Microtia and congenital meatal atresia are due to maldevelopment of the foregoing structures. This maldevelopment must occur in the second month of fetal life; not only are the auricle and external meatus and tympanum malformed, but also the malleus and incus, which arise from the cartilaginous bar of the first arch. The malleus is especially prone to malformation. Further, the laterohyal, which is formed from the cartilaginous bar of the second arch, is usually excessively developed and, in many cases, is responsible for the bony meatal atresia. The cause of the malformation is probably a fault in germ plasma, but in rare cases intra-uterine injury may account for a mild deformity of the auricle.

"3. The tubercles that form on the external surface of the first or mandibular arch do not develop in the normal manner, and several varieties of auricular deformity are described, varying from slight cases, such as pointed or darwinian ear, to total absence of the auricle.

"4. The invagination of the surface epithelium which forms the primitive external acoustic meatus fails to develop or does so imperfectly, and in consequence the tympanic ring, which is formed by an ossification of the mesoblast around the tympanic membrane, is congenitally defective or even absent. The meatal atresia may be membranous or bony.

"5. The otic vesicle, from which the membranous labyrinth is formed, develops earlier than, and quite independent of, the external and middle ears. The labyrinth is usually normal in cases of microtia and congenital meatal atresia.

"6. In a typical case the auricle is malformed or absent, the external meatus is occluded by bone, the malleus is small or absent, while the incus is large but misshapen. The tympanum is narrowed, not only from above downward, but also from side to side, and the window niches are occluded by connective tissue. The facial nerve is usually small and may cross the tympanum uncovered by bone.

"7. Clinically, the condition of microtia and meatal atresia is not rare. It occurs more frequently in males than in females and more often on the right side than on the left. Unilateral cases are more common than bilateral. In rare instances the condition occurs in more than one member in a family. Facial paresis, maldevelopment of the mandible, facial hemiatrophy and other congenital deformities are not infrequently noted in association with microtia and meatal atresia.

"8. Hearing tests, as might be expected, usually show the results obtained in lesions of the sound-conducting apparatus, but even in bilateral cases the human voice is, as a rule, heard well enough to allow the patient's speech to develop (Toynbee). Functional examination by means of the rotation tests shows, as a rule, that the vestibular apparatus is normal.

"9. Operation should be performed in unilateral cases only when otitis media and mastoiditis are present on the affected side. In other unilateral cases the improvement in hearing is not sufficient to justify operation (Alexander). In bilateral cases, operation is called for only if the deafness is marked and if roentgenograms and functional examinations show that the labyrinth is healthy (Marx)."

BARNETT, Chicago.

ARE TONSILLAR RECURRENCES ENTIRELY DUE TO FAULTY OPERATIVE TECHNIC? NORMAN LESHIN and SAMUEL J. PEARLMAN, Arch. Otolaryng. **13**:37 (Jan.) 1931.

Many recurrences of tonsils following operation are the result of poor operative technic, but at least 50 per cent of recurrences cannot be attributed to this cause.

The plicae of the tonsils are three extensions of the so-called capsule. They have much to do with the original formation of the tonsil, and microscopic study of them after tonsillectomy shows small lymphoid structures that can and often do develop into new tonsillar tissue. The plicae may or not be removed during

operation; this is rarely done by the guillotine method. Further, there is no operative method that insures the patient positively that there will not be lymphoid growth at the site of operation because even in the tunica propria of the mucosa surrounding the fossae lymphoid tissue has been found that is capable of hypertrophy, and this is carried into the fossae as the mucosa extends to epithelialize the raw operative surface.

Just why some persons will have hypertrophy of these lymphoid structures following operation and others will not is not known.

BARNETT, Chicago.

PRESENT-DAY METHOD FOR TESTING THE HEARING AMONG SCHOOL CHILDREN. EMILY A. PRATT, New York State J. Med. **31**:81 (Jan. 15) 1931.

The Medical Inspection Bureau of the State Department of Education is urging the general use of the phono-audiometer for testing the hearing of all school children. Tests were made in several cities of the state; 18,198 children were tested; 6.4 per cent gave evidence of defective hearing; 54 per cent of these deaf children were found to be backward in school, being one or more grades behind the average.

AIKMAN, Rochester, N. Y.

THE TONSILS AND NASOPHARYNGEAL EPIDEMICS. W. H. BRADLEY, Arch. Dis. Childhood **5**:335 (Oct.) 1930.

Two hundred and eighty-nine boys, in good health, were examined during July, 1929, and data relative to the tonsil, the oral pharynx, the buccal mucosa and the cervical gland were noted; in 167 of these the tonsils had been removed previously.

The same group was observed for one year for the incidence of catarrhal disease. Removal of the tonsils and adenoids was found to be of no appreciable value in the prevention of epidemic nasopharyngeal infection. There is some evidence to show that it is followed by compensatory hypertrophy of other lymphoid tissue in the presence of disease of the upper air passages. The observations suggest that until the loss of lymphoid tissue is made good the patients operated on are more susceptible to common catarrh and more frequently attacked by complications.

Patients with chronic tonsillitis who are subject to recurrent auto-infection are rare, and their number is insufficient to alter this conclusion, disease usually being due to transitory surface infection. The quality of lymphoid tissue has not been investigated. There is, however, considerable evidence than an optimum quantity exists in the healthy throat, and that this optimum quantity is greatest in the presence of nasopharyngeal infection. Reduction of this quantity by operation increases the patient's risks to invasion by an infecting organism, not only so far as simple catarrh is concerned but to a greater extent with regard to complications.

Palpable cervical glands, even when enlarged, are seldom pathologic but are most frequently physiologic. This physiologic enlargement results from the presence of infection in the nasopharynx, which also stimulates growth of the nasopharyngeal lymphoid tissue, the tonsil or the tonsillar remnant. The increased risk of invasion after operation is in some way mitigated by this compensatory hypertrophy of the remaining lymphoid tissue, and the absence of hypertrophy appears to conduce to complications. The distinction between infection and invasion should be noted. When a pathologic flora attacks a closed community, almost every member becomes infected and the carrier rate is said to be very high. Comparatively few of the infected hosts respond with a local and general reaction sufficiently violent to constitute disease. The latter are said to be invaded. It has also been suggested that the common cold is the expression of an abortive reaction to infection, no matter what the infecting organism. The continued search for a specific cause of the common cold is not likely to establish a single pathologic process for this condition.

That most extensive and ubiquitous group of diseases, the droplet infections, has little to do with the tonsil or its removal. Except in the relatively rare instance in which auto-infection from a chronic focus produces recurrent inflammation, acute tonsillitis is a symptom and not a disease. Tonsillectomy is symptomatic treatment in its most elementary form, and its extensive application has produced no obvious beneficial result in the control of acute nasopharyngeal infections.

KELLY, Milwaukee.

REPORT ON TWO CASES OF SEPTICAEMIA FOLLOWING TONSILLECTOMY. W. R. F. COLLIS, Proc. Roy. Soc. Med. **24**:318 (Jan.) 1931.

Case 1: A girl, aged 5 years, had her tonsils removed by enucleation. A small peritonsillar abscess was found at operation; otherwise nothing unusual was noticed. The following day a high temperature commenced, and the child died with all the symptoms of septicemia the next day.

Case 2: A girl, aged 7 years, was operated on after the child in case 1, on the same day. It was noticed at operation that the follicles were inflamed and the tonsillar glands enlarged. The operation was performed without incident, and the following day all the symptoms of septicemia developed with a high swinging temperature, rigor, and the presence of blood and albumin in the urine. The child died. Both children's blood showed *Streptococcus scarlatinae*. The children lay next each other in the recovery room. The father and sister of the second patient had contracted scarlet fever about the same time. Swabs were taken from their throats, and the presence of the same type of scarlet fever streptococcus as that found in the heart's blood in case 1 was demonstrated.

WILLIAMSON, New Orleans.

TREATMENT OF THE DEAF INFANT. G. DE PARREL, Progrès méd., Dec. 13, 1930, p. 2208.

The author classifies deaf infants in three groups: deaf-mutes, partially deaf children and those hard of hearing. The deaf-mutes should be given over to educational specialists at 3 years of age. The partially deaf frequently show rhinolaryngologic defects and should be examined thoroughly, after which special education is indicated. The hard of hearing should be in special classes of not over eight or ten pupils.

SANFORD, Chicago.

NINE CASES OF OTOGENOUS ABSCESS OF THE BRAIN. A. REJTÖ, Monatschr. f. Ohrenh. **64**:1174 (Oct.) 1930.

In nine patients with otogenous abscess observed by Rejtö, the temporal lobe was involved; sometimes the abscess extended to the occipital lobe. Eight of the patients were males and one a female; their ages ranged from 7 to 42. In three instances, surgical intervention took place as soon as signs of cerebral abscess developed; the patients recovered. The other six patients were admitted to the hospital with a pronounced meningitis; all of them died. Four patients were observed for from sixteen to twenty-eight days before signs of a cerebral abscess became manifest. In one case general tuberculous symptoms prevailed at first; at the fourth week an ataxic gait and incoordinated movements of the arm appeared, pointing to a cerebral abscess. In another case, mental disturbances were the predominant feature in the beginning; later, sudden appearance of a severe headache and slowing up of the pulse suggested the diagnosis of a cerebral abscess. In a third patient, severe headache, slowing up of the pulse and sudden diplopia established the diagnosis of cerebral abscess; in the fourth case, a slow pulse, ataxic speech, spontaneous nystagmus and incoordination of movements of the hand confirmed the diagnosis of a cerebral abscess. All patients admitted in the manifest stage presented meningeal signs. In these cases, the dura mater of the middle cranial fossa was exposed; puncture of the dura confirmed the presence

of an abscess. In the nine cases, the suppuration was chronic; there was an acute exacerbation of the chronic otitis in seven. The acute exacerbation was characterized by headache, pain in the ear, also frequently by fever; congestion of the tympanic membrane and of the tympanic cavity, sometimes tenderness or swelling of the mastoid process were found. The acute exacerbation was associated with acute nephritis in one instance and with endocarditis in another. Cerebral abscess may develop from a contact infection, whereby lesions of the bone lead to meningitis, or from encephalitis caused by a retrograde septic thrombosis in the veins of the middle ear. In the three patients who recovered, the abscess developed by the venous route; it was located in the white substance. In one of these cases Lemaitre's operation and drainage were used; in another the abscess was incised and iodoform gauze was inserted. The tampons were first changed daily, for eight successive days; then four times, at intervals of a day. In the third case, the tampons were renewed only twice. There was evidence that the results depended more on the patient's condition at the time of the operation and on the strength of his resistance than on the surgical method used.

EDITOR'S ABSTRACT [ARCH. OTOLARYNG.]

OTITIS AND MASTOIDITIS IN NURSLINGS. G. ALEXANDER, Monatschr. f. Ohrenh. **65**:437 (April) 1931.

Alexander begins his article with a discussion of the peculiarities of otitis and mastoiditis in nurslings. He points out that there are anatomic, physiologic and clinical differences. He then discusses the diagnosis and treatment. In the conclusion, he stresses the following points: 1. In all nutritional disturbances of nurslings the ears should be examined regularly, whether or not symptoms are present. 2. Inflammatory hyperemia of the tympanic membrane can often be recognized on the fine red branches in a gray opaque field. 3. Lowering of the posterior upper wall of the auditory meatus indicates mastoiditis or antritis. 4. Edema above the mastoid process can be detected only by comparative palpation of both mastoids. 5. If the otorrhea has continued for four weeks without showing a tendency to heal (no slimy pus), the mastoidea should be opened irrespective of local signs. 6. In cases of bilateral suppurative otitis paracentesis should be performed immediately and simultaneously on both sides. In cases of bilateral mastoiditis, however, the most severely affected side should be opened first; the other side often heals without operative intervention. The operative trauma caused by a bilateral mastoidotomy that is performed in one session is too severe for a nursling. 7. Owing to the difficulty of otoscopy in nurslings, the amount of latent otitis increases. Really latent are the tuberculous otitis and tuberculous mastoiditis which occur comparatively frequently in infants up to 8 months of age. 8. Under poor hygienic living conditions, the dangerous combination of nasopharyngeal otitis and dyspepsia increases in frequency and in severity.

EDITOR'S ABSTRACT [ARCH. OTOLARYNG.]

HEREDITARY AND CONSTITUTIONAL PATHOLOGIC CONDITIONS OF EAR AND OF UPPER RESPIRATORY PASSAGES. SIEMENS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:3 (May 5) 1931.

Siemens first reviews the foundations of the modern theory of heredity. He explains homozygoty and heterozygoty, dominant and recessive heredity, and sex limited and sex bound heredity. Further, he stresses the difficulties in researches on hereditary pathologic conditions, and points out how insufficient knowledge may lead to grave errors. He deplores that investigations on heredity are still based largely on guess work. Genealogical trees are mostly built on the basis of anamnestic reports, which naturally are not always reliable, for the same pathologic character may be due to different hereditary "anlagen." Deaf-mutism, for instance, may be idiotypic, namely, recessively hereditary; it may be one aspect of cretinous degeneration, or the result of intra-uterine syphilis, or it may be acquired by birth trauma, by traumatic injury or by labyrinthitis during the nurslings

period. This indicates that the various etiologic groups have to be differentiated. The author also warns against the overemphasis on positive cases and stresses that the material on which hereditary studies are made should be large enough. He further points out that not only one pathologic character may be caused by several anlagen, but that one anlage may cause several pathologic conditions. This so-called polyphenic heredity exists, then, when certain diseases are passed on in more or less definite correlation, like osteopsathyrosis for instance. In this case a certain dominant anlage not only transmits the fragility of the bones but also the blue color of the scleras, and later in life there often develops a special form of otosclerosis. To such cases, in which not only one certain organ is affected, the term constitutional disease is frequently applied. The author criticizes the commonly accepted definitions for constitution and points out that the term constitutional disease generally denotes correlations of diseases, and he therefore agrees with Kraus who defines constitution in the sense of syzygiology or correlation. By means of careful statistical methods, one should be able to determine how often and under what conditions otosclerosis develops in osteopsathyrosis, or how great is the certainty that children with hyperplastic pharyngeal ring ("adenoid diathesis") will develop pharyngeal catarrhs or otitis media, or how often certain anomalies of the ear concur with mental disorders and imbecility. As yet little definite knowledge exists concerning correlation. In otology, the frequency of correlations, and consequently the significance of constitution, are often overvalued. All methods that are employed in hereditary studies are based in principle on the determination of the frequency of characteristics in certain spheres of relationship. Such spheres of relationship are the family, the race and twinship. The author evaluates these three methods. Racial pathologic characteristics are of minor value in hereditary research because the different races generally live in a different environment and in a different social milieu; thus it is difficult to determine whether differences in the disease frequency of two races is the result of heredity or of other factors. It is also known that marriages between blood relatives lead to a more frequent manifestation of pathologic conditions, but this practice varies in different races. Investigations on twins are no doubt the most reliable method for the study of hereditary pathologic characteristics. They are particularly helpful in checking the results of familial pathologic studies. In the conclusion, the author points out that the aim of researches on hereditary pathologic conditions should not only be to increase our knowledge about hereditary diseases, but that this knowledge should be a means to a higher aim. A causal therapy in hereditary diseases should be elimination of the pathologic anlagen, but care should be taken that the healthy anlagen are retained.

EDITOR'S ABSTRACT [ARCH. OTOLARYNG.]

THE SEDIMENTATION OF ERYTHROCYTES IN INFECTIONS OF THE MIDDLE EAR. V. HLAVÁČEK, Časop. lék. česk. **67**:1395 (Oct. 5) 1928.

Cases of mastoiditis showed increased sedimentation of red cells independent of temperature. Extensive lesions were found in all cases of accelerated sedimentation. The figures for increased sedimentation were comparable with those for extensive pathologic processes, such as tuberculosis, with marked elevation of temperature. Practically only the decreased sedimentation time is of value with other signs and symptoms in deciding as to operation. In cases of decreased sedimentation no tissue detritus except nonpurulent cholesteatoma was noted.

STULIK, Chicago.

STATISTICAL REPORT ON THE CAUSE OF DEAF-MUTISM IN THE VYMOLA INSTITUTE IN PRAGUE. VÝMOLA, Časop. lék. česk. **67**:1525 (Nov. 2) 1928.

In 112 patients, the diagnosis on objective indications was congenital deaf-mutism in 71, acquired deaf-mutism in 37 and deaf-mutism of undetermined origin in 4. On careful checking of the family history it was found that the condition

was truly congenital in only 53. The reasons cited for acquired deaf-mutism were: undetermined fever, 2 cases; pertussis, 1 case; grip, 2 cases; meningitis, 1 case; otitis media, 1 case; measles, 1 case; convulsions, 3 cases; trauma (fall), 4 cases; fright, 2 cases, and difficult teething, 1 case. The causes given in the series cited are: consanguinity, mendelian heredity, mongolism, constitutional diseases, alcoholism, infectious diseases and social environment.

STULIK, Chicago.

Diseases of the Skin

FAVUS WITH LESIONS ON THE GLABROUS SKIN. J. GATÉ, G. MASSIA and TREPPOZ, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Lyon) **38**:146 (Jan.) 1931.

In an infant, aged 2 years, favus of the scalp with generalized pityriasiform lesions were present. Scales from the latter cultivated and examined under the microscope showed *Achorion*. In the preparation of the scales, *Mycelia* resembling *Microsporon* were found, but the cultures never showed fungi of the *Trichophyton* type. The authors call special attention to this condition, since error may occur if cultures are not made.

MICHAEL, Houston, Texas [ARCH. DERMAT. & SYPH.]

DUHRING'S DISEASE IN A CHILD WHOSE MOTHER HAD TERTIARY SYPHILIS. SPILLMANN, WEIS and SEGALL, Bull. Soc. franç. de dermat. et syph. (Réunion Nancy) **38**:355 (March) 1931.

The case recorded is that of a boy of 4 years who had a typical dermatitis herpetiformis. The mother had tertiary syphilis. The child, who had no clinical or serologic evidence of syphilis, was given sulpharsphenamine with disappearance of the eruption in about one month. The authors report the case without implications.

MICHAEL, Houston, Texas [ARCH. DERMAT. & SYPH.]

VARIATIONS IN THE ACID-BASE BALANCE IN SOME DERMATOSES. SPILLMANN, VERAIN and WEISS, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Nancy) **38**:364 (March) 1931.

While admitting the importance of the conception of allergy in the etiology of eczema, the authors do not believe that all cases of that disease are of allergic origin. For some years they have been studying the acid-base balance in several diseases (eczema, prurigo, urticaria, psoriasis) and reported their results in the *Annales de dermatologie et syphiligraphie* (**10**:1301 [Dec.] 1929). In the present report they record examinations in 116 new cases. They employed an electrometric measurement of the blood p_H.

Of 80 cases of eczema, 60 showed alkalosis, 4 normal figures and 16 acidosis. Of 16 cases of psoriasis, 12 showed alkalosis, 1 normal figures and 3 acidosis. Of 3 cases of urticarias, 2 showed acidosis and 1 alkalosis. Four cases of dermatitis seborrheica showed alkalosis and 1 acidosis. Five cases of cutaneous pruritus and prurigo showed alkalosis and 2 acidosis. One case of Duhring's disease gave alkalotic figures, the other acidotic. Two cases of erythroderma (not qualified) showed alkalosis and 1 acidosis.

From this study, it is concluded that an acid-base imbalance is present in certain skin diseases that are regarded as cutaneous reactions. It plays an especially prominent rôle in those having an exudative feature (eczema particularly). The imbalance may be toward either the acid side (22 per cent) or the alkalotic (73 per cent). The degree of imbalance varies with the case rather than with the intensity of the eruption, except that in each case there appears to be a close relation between fluctuations in the acid-base dysequilibrium and the clinical course. There were, however, some exceptions to this general rule. From the therapeutic

standpoint, those cases showing acidosis were most amenable to therapy directed toward the p_H imbalance. The alkalotic cases were less influenceable.

In general, it may be concluded that an acid-base disequilibrium is one of the features of a dermatopathic terrain. It is not a question of whether there is an acidosis or alkalosis but whether there is a derangement of the acid-base balance.

MICHAEL, Houston, Texas [ARCH. DERMAT. & SYPH.]

APLASIA MONILIFORMIS. L. M. PAUTRIER and R. GLASSER, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:305 (March) 1931.

The unusual feature about this otherwise typical case in a child of 11 years was that it was the first occurrence of the disorder in the family.

MICHAEL, Houston, Texas [ARCH. DERMAT. & SYPH.]

THE PATHOGENESIS AND ETIOLOGY OF ERYTHEMA NODOSUM. GEDEON DÓCZY, Dermat. Wchnschr. **92**:241 (Feb. 14) 1931.

The author briefly reviews some of the recent literature on erythema nodosum and concludes that there is no single etiologic explanation for this condition. In his study of eight cases he finds an instability of the sympathetic nervous system and also notes a tendency toward acidity of the urine.

TAUSSIG, San Francisco [ARCH. DERMAT. & SYPH.]

A CONTRIBUTION TO THE KNOWLEDGE OF "ATROPHODERMIA VERMICULATA." L. LEVEN, Dermat. Ztschr. **60**:250 (Feb.) 1931.

A patient is described with typical atrophodermia vermiculata which had been present since birth. This was not accompanied at any time by the formation of comedones or by any inflammatory manifestation. The literature is briefly reviewed and the author concludes that there are two groups: one associated with folliculitis and comedones, which should be classified as folliculitis ulerythematosa reticulata, acne vermoulante or ulerythema acneiforme; the other, a noninflammatory condition, which should be called atrophodermia vermiculata. The latter probably belongs to the nevus group.

TAUSSIG, San Francisco [ARCH. DERMAT. & SYPH.]

Surgery and Orthopedics

SPLENECTOMY FOR HEMORRHAGIC PURPURA OF CHILDREN. FRED W. RANKIN and RICHARD S. ANDERSON, Ann. Surg. **93**:749 (March) 1931.

Because the removal of any organ from an extremely young child is a formidable procedure, the authors report the case of a child aged 4 years, in whom the spleen was removed for hemorrhagic purpura. In reviewing the literature the authors found two other patients, slightly younger, who had had the spleen removed with favorable results. The authors give in detail the laboratory observations before and after operation. The improvement, as shown in the laboratory from one week prior to operation to two weeks afterward, is briefly as follows: The platelets increased from 30,000 to 84,000 and the hemoglobin from 53 to 60 per cent; the erythrocytes were reduced from 3,930,000 to 3,500,000, coagulation time from 14 to 5 minutes, and bleeding time from 40 to 60 minutes to 10 to 20 minutes.

In discussing the diagnosis the authors state that hemorrhagic purpura indicates the spontaneous extravasation of blood into or under the skin and mucous membrane of the body. It is only a symptom and its etiology is uncertain. The important indications in the blood are: (1) increased bleeding time, (2) a decreased platelet count, (3) a nonretractile clot and (4) normal or slightly increased coagulation time. There is a positive reaction to the Rumpel-Leeds test. This test is carried out by applying a blood pressure cuff to the arm at the mean blood pressure for five minutes. The test is positive if, within a few

minutes after removal of the cuff, purpuric spots appear on the arm below the cuff. Profuse spots and spots of large size are almost pathognomonic of hemorrhagic purpura.

The treatment advised is splenectomy. The earlier the operation is performed in the course of the disease, the better the results are said to be. If the platelet count is extremely low, and the bleeding time is much prolonged, one is advised to give preoperative treatment, such as transfusions and intravenous injections of calcium. Forty patients on whom splenectomy was performed at the Mayo Clinic are living and in good health with no tendency to bleeding.

BANTIN, Omaha.

THE SURGICAL TREATMENT OF SOME CONGENITAL ABNORMALITIES OF THE GENITO-URINARY TRACT. WALTMAN WALTERS, Ann. Surg. **93**:862 (April) 1931.

The author states that the most frequent defects and abnormalities of the genito-urinary tract are hydronephrosis, undescended testicles, exstrophy of the bladder, hypospadias and epispadias. In discussing the treatment for hydronephrosis, he emphasizes the benefits from conservative operation. In the treatment for exstrophy of the bladder and complete epispadias, with absence of the urinary sphincters, he advocates transplantation of the ureters into the rectum and the removal of the malformed bladder. The undescended testicle, he states, should be brought down and placed in the scrotum before the age of puberty, so that it may grow normally and function satisfactorily. In treating patients for hypospadias and epispadias, he obtained his best results in children between 6 and 9 years of age. He discusses in detail the surgical treatment for hydronephrosis, exstrophy of the bladder and undescended testicles.

BANTIN, Omaha.

THE SURGICAL ASPECTS OF ASCARIASIS. EARLE I. GREENE and J. MAJOR GREENE, Ann. Surg. **93**:920 (April) 1931.

The authors state that ascariasis is rarely thought of in considering diseases that require surgical intervention. *Ascaris lumbricoides* is famous for its habit of wandering about and making its appearance in strange places. At times it may produce grave pathologic states. If these serious disturbances are borne in mind, more examinations of the stool will be made and some of the complications prevented. The authors report various surgical conditions found in children due to ascariasis, such as intestinal obstruction, perforation of the bowel, pancreatic disease, disease of the gallbladder, appendicitis and empyema with pulmonary gangrene.

BANTIN, Omaha.

INTUSSUSCEPTION IN AN INFANT WITH UNUSUAL PHYSICAL FINDINGS. SAMUEL GOLDBERG, Arch. Pediat. **48**:135 (Feb.) 1931.

The author reports a case of intussusception in an infant aged 10 months. A characteristic history and picture were noted, but the abdominal examination gave entirely negative results.

WADDELL, University, Va.

PERITONITIS: THE CORRELATION OF THE BACTERIOLOGY OF THE PERITONEAL EXUDATES AND THE CLINICAL COURSE OF THE DISEASE IN ONE HUNDRED AND SIX CASES OF PERITONITIS. FRANK L. MELENEY, H. D. HARVEY and H. Z. JERN, Arch. Surg. **22**:1 (Jan.) 1931.

The great majority of patients with peritonitis have lesions of the appendix. The severity of the case depends on whether the appendix has perforated or not. A diffuse peritonitis was always more severe than when abscesses had formed and the mortality was twice as high. Smears made at the time of operation were

of prognostic value. When smears showed no organisms and cultures yielded no growth, or when fewer species appeared in culture than were seen on the smear, every patient recovered. When more kinds of organisms appeared on culture than were seen on the smear, there was an indication that certain organisms were of potential danger. When all of the forms seen on the smear grew out, the mortality was still higher, which warranted a guarded prognosis. In this series peritonitis was usually a polymicrobic disease. The lowest mortality was in patients under 20 years of age. The author concludes that bacteriologic studies are of great prognostic value.

KAISER, Rochester, N. Y.

ATTITUDES ASSOCIATED WITH LESIONS ABOUT THE HIP JOINT. VERNON L. HART, Arch. Surg. **22**:463 (March) 1931.

The author points out that one can frequently gain considerable information from a careful study of the position of deformity or attitude of the lower extremities associated with lesions involving the hip joint. To make a correct interpretation of the position of deformity of an extremity, it is necessary to place the pelvis on a transverse and horizontal axis. The "frog" attitude in a child should lead the physician to suspect one of the pseudoparalyses associated with rickets, scurvy or syphilitic epiphysitis. The position of flexion, adduction and internal rotation known as "the position of instability" is productive of spontaneous dislocation and should be considered a dangerous attitude.

KAISER, Rochester, N. Y.

THE CLINICAL RESULTS FOLLOWING THE OPERATION OF SYMPATHETIC RAMISECTION. N. D. ROYLE, Canad. M. A. J. **24**:229 (Feb.) 1931.

According to the author, the chief indication for sympathetic ramisection is the failure of inhibition in voluntary muscles. The operation decreases tone and thus enables patients to move smoothly and more rapidly. The author has performed the operation approximately 600 times, and only 3 deaths have occurred.

In congenital spastic paraplegia the following results were noted in 37 cases: excellent in 8 (21.62 per cent), good in 20 (54.05 per cent), poor in 5 (13.51 per cent) and no results at all 4 (10.80 per cent). In 10 patients with congenital spastic chorea the results were: excellent in 3 (30 per cent), good in 4 (40 per cent) and poor in 3 (30 per cent).

The method was used in 5 cases of chronic constipation and megalocolon in adults, but no mention is made of any cases of this disorder in children.

The results in acquired spastic paraplegia, parkinsonian rigidity, sarcoma of the femur, writers' cramp, conditions simulating tic douloureux, various vasospastic conditions and retinitis pigmentosa are given.

MOORE, Omaha.

SYMPATHECTOMY FOR MEGALOCOLON. D. E. ROBERTSON, Canad. M. A. J. **24**: 359 (March) 1931.

This is a report of three cases of megacolon, congenital in origin. The patients were treated by surgical intervention on the lumbar sympathetic trunk. In the approach to the lumbar sympathetic the technic was that advocated by Royle, the details of which are given by the author. In two of the cases the left branches of the sympathetic system were severed and in one case both the right and the left branches were divided. Following the operation the foot on the affected side became dry and warm. All of the cases showed definite improvement. In one there was a mild relapse which cleared up after the use of enemas. Roentgenograms showed a definite decrease in the size of the colon with evidence of increased spasticity. Since this report the author has had three additional cases of megacolon which were cured by sympathectomy.

MOORE, Omaha.

TREATMENT OF PULMONARY ABSCESSES WITH INTRATRACHEAL INJECTIONS. N. A. ILJASCHENKO, Monatschr. f. Ohrenh. **64**:1340 (Nov.) 1930.

In one of Iljaschenko's patients, a man, aged 42, an abscess developed in the inferior pulmonary lobe on the right side. During the six months of treatment, the patient had been given eleven intratracheal injections of Besredka's antivirus, each dose containing from 30 to 60 cc., fifty-seven intratracheal injections of from 10 to 20 cc. of a 6 per cent oily solution of guaiacol and eucalyptol, and thirty-two intratracheal injections of from 20 to 25 cc. of a 5 per cent water solution of thiocol (once 50 cc. was injected). In addition, five intravenous injections of a 25 per cent solution of dextrose were given; each of three of the injections contained 10 cc. and each of two injections contained 20 cc. The fever almost disappeared, and the daily amount of mucus decreased from 450 to 60 cc.; the cough became notably reduced, and the general condition improved. Another patient, aged 50, presented an abscess involving the middle and the inferior lobes of the left lung. The treatment was continued for about six weeks; it consisted of fifteen intratracheal injections of from 10 to 20 cc. of guaiacol and eucalyptol and one injection of thiocol. This treatment was followed by the disappearance of the odor from the mouth; the temperature became almost normal, and the mucus raised was reduced from 300 to 100 cc. a day. There was relief from the cough, and general improvement resulted. The third patient, a youth, aged 19, suffered with pulmonary tuberculosis and an abscess in the right lung. During the first three months of treatment, he was given five intratracheal injections of from 15 to 25 cc. of antivirus, ten injections of from 5 to 20 cc. of guaiacol and eucalyptol, nineteen injections of from 20 to 30 cc. (once 60 cc.) of thiocol and seven intravenous injections of 20 cc. of dextrose. The treatment was continued for the next five months with two intervals, each of a month's duration. During the second series of treatment, forty-three intratracheal injections of from 10 to 25 cc. of guaiacol and eucalyptol were given. The fever subsided, the cough was considerably diminished; the mucous discharge was reduced, and the subjective signs improved. In a fourth patient, aged 51, the abscess was situated in the inferior lobe of the left lung. Three intratracheal injections of from 15 to 20 cc. of antivirus, thirty-one injections of from 10 to 50 cc. of guaiacol and eucalyptol and two intravenous injections of 10 cc. of dextrose, were given within the first seventy days of treatment. Twenty-four intratracheal injections of from 25 to 50 cc. of thiocol, as well as fifteen injections of from 20 to 50 cc. of guaiacol and eucalyptol, were given within the next seventy days. The cough almost disappeared; the amount of sputum became insignificant, and the fetid odor disappeared from the mouth. The patient gained 21 pounds (9.5 Kg.) in weight. In all cases the clinical improvement was confirmed on roentgenologic examination, indicating that a fibrous tissue had developed in the region previously occupied by the abscess. The intratracheal injections were well tolerated. The author stresses the fact that no decisive conclusions can be drawn as long as the treatment has not been tried in a large number of patients.

EDITOR'S ABSTRACT [ARCH. OTOLARYNG.]

THE CLINICAL DIAGNOSIS OF DIAPHRAGMATIC HERNIA IN INFANTS. C. BONZANIGO, Ztschr. f. Kinderh. **50**:253, 1930.

Up to 1912, only 15 of 650 cases of diaphragmatic hernia reported in the literature were recognized during life; from 1912 to 1929, 27 of 81 cases were recognized, only 6 by clinical observation. A child 5½ years of age did not thrive; it became dyspneic easily, and cyanotic on crying. The breathing was difficult and not rapid; moderate respiration was accompanied by a drawing in of the intercostal spaces on both sides. The thorax was distended throughout but more so in the left lower portion. The entire left half was dull, and the breath sounds feeble. Marked dextrocardia was observed. The diagnosis was made by roentgen examination. By daily clinical observation, the sound was

seen to change; it was dull on some days, breathing being feeble to absent; on others the sound was tympanitic, with amphoric breathing. There was a metallic murmur not dependent on the breathing but on the movements of the intestine.

EMERSON, Boston.

Miscellaneous

A QUANTITATIVE STUDY OF BEHAVIOR PROBLEMS IN RELATION TO FAMILY CONSTELLATION. JOHN LEVY, Am. J. Psychiat. **10**:637 (Jan.) 1931.

This report is based on a study of 700 children presenting behavior problems; in all cases the intelligence quotient was 80 or above, so that the factor of mental deficiency was avoided. An effort was made to determine, according to the size of the family, the distribution of problem children in the clinic group and the distribution of children in the general population of Chicago. The distribution of problem children and controls with respect to the economic status of the parents was also analyzed as was the economic status of the parents in the general population of Chicago. An attempt was then made to study the behavior of children with reference to the size of the family in a small rich community, and to compare the distribution of problem children in a rich community with reference to the size of the family. The statistics also included such problems as only children, first-born children, second-born, etc.

Analysis of these data shows that behavior problems are independent of the size of the family. In the small rich community the only child more frequently presents problems of behavior than children in larger families. This is true chiefly of boys. Only girls seldom become problems in the small, well-to-do community. In a large city boys develop behavior difficulties more than twice as often as girls. In a large city the first-born child is a problem more frequently than children in any other ordinal position. In a high grade community behavior difficulties develop in the second-born child more frequently than in children in other ordinal positions. The only child does not appear to be a "spoiled child" as frequently as children from two children families. Only children are troubled much more by scholastic difficulty than are children of larger families.

JAHR, Omaha.

BLOOD TRANSFUSIONS IN CHILDREN. PHILLIP E. ROTHMAN, California & West. Med. **34**:176 (March) 1931.

Transfusions are advocated in the anemias of childhood, in the early stages of acute infections and in pneumonia, erysipelas, marasmus, burns, chronic sepsis and other conditions. It is safe to employ any method that can be used skilfully. The rate of 10 cc. per minute is the proper one, the amount ranging from 10 to 40 cc. per kilogram. Other factors, such as the careful matching of the blood of the donor and the recipient, are discussed.

DICKEY, San Francisco.

SLEEP AND DISORDERS IN CHILDHOOD. HECTOR CHARLES CAMERON, Canad. M. A. J. **24**:239 (Feb.) 1931.

The subject of disorders of sleep in childhood is discussed from a purely clinical standpoint. Four types of this disturbance are considered: (1) sleeplessness and continuous crying in young infants, (2) sleeplessness in older children, (3) night terrors and (4) enuresis.

Three factors are concerned in the production of the first type: 1. Pain or discomfort. The most common causes for this are colic, particularly that due to aerophagy and to nasal obstruction. Various other causes are mentioned, such as syphilitic periostitis, subperiosteal hemorrhages of scurvy, uric acid calculi and otitis media. 2. Constitutional neuropathy. This is a rather infrequent cause of sleeplessness. 3. Faulty management. This is a common cause.

Sleeplessness in older children is frequently psychic in origin, but in a given case a review of all the circumstances of the child's life should be made in the search for contributory causes. In the nervous type of child the relative nocturnal hypoglycemia and ketonemia may be a factor in the production of night terrors and enuresis.

The unstable metabolism of the child, with the tendency toward the production of hypoglycemia and ketonemia, is discussed in some detail.

MOORE, Omaha.

PROBLEMS IN CHILD DEVELOPMENT. A. B. CHANDLER, Canad. M. A. J. **24**:404 (March) 1931.

This report is based on a study of one hundred cases in the Development Clinic of the Child Welfare Association of Montreal. The clinics are held once a month in four of the health centers at widely separated portions of the city; all cases must be referred by the physician or nurse of the health center. The ages of the children varied between 6 months and 6 years. Physical examinations gave almost entirely negative results, except for slight defects of posture, teeth and tonsils, such as one would find in any group of children of this age. A careful study of the environment is of the greatest importance. Carefully elicited information as to the child's daily life, the mother's reaction to the difficulties that she sees in its behavior and an accurate idea of the essential points in its character help greatly in finding a solution for these types of trouble.

MOORE, Omaha.

SHOE-DYE POISONING. S. J. LEVIN, J. A. M. A. **96**:681 (Feb. 28) 1931.

In an infant, aged 8 months, shoe-dye poisoning developed one hour after dyed shoes were placed on its feet. A 20 per cent solution of orthotoluidine, which was used as a solvent in this shoe dye, was as toxic as the aniline or nitrobenzene solvents formerly used.

BONAR, Salt Lake City.

PEDIATRIC REFLECTIONS. JOSEPH H. MARCUS, M. J. & Rec. **132**:484 (Nov. 19) 1930.

The author gives a few reflections on the following subjects: (1) respiratory and gastro-intestinal disorders, (2) impetigo, (3) cough, (4) premature infants, (5) mastoid involvement, (6) atropine poison, (7) retropharyngeal abscess, (8) tuberculous pleurisy and (9) the feeding of infants.

WILLIAMSON, New Orleans.

THE MOTHERS' MILK BUREAU OF THE CHILDREN'S WELFARE FEDERATION. HENRY DWIGHT CHAPIN, M. J. & Rec. **133**:53 (Jan. 21) 1931.

The Mothers' Milk Bureau of the Children's Welfare Federation was started in 1921. The collection of milk is generally from Italian mothers referred to the Bureau by infant health stations and postnatal clinics. The method of collection, technic and sale of the milk are described.

WILLIAMSON, New Orleans.

IRRADIATION OF SCHOOL CHILDREN. LEONARD HILL and ALAN R. LAURIE, Lancet **1**:182 (Jan. 24) 1931.

Four groups of 100 children each were studied. Group 1 were irradiated with a long flame tungsten-cored carbon arc twice weekly. Group 2 were given a pellet containing the vitamin D equivalent of 2 ounces (59 Gm.) of cod liver oil daily. Group 3 were given similar looking pellets containing no irradiated ergosterol and were exposed to an arc fitted with a glass screen. (This group was studied to see what part psychology plays in the taking of pellets and undressing before a lamp.) Group 4, the controls, were untreated. The test extended over sixteen weeks, and groups 1 and 2 showed marked improvement by

an appreciable margin. The children who made the spectacular improvement were in group 1. The greatest effect seemed to be on their eating and sleeping.

LANGMANN, New York.

THE RATIO OF CIRCUMFERENCE OF THE CHEST TO HEIGHT IN TALL CHILDREN. P. NOBÉCOURT, Gaz. d. hôp. **104**:361 (March 7); 397 (March 14) 1931.

In a previous paper the author published the measurements of the chest in 215 tall children and in his present publication he calculates the ratio of the circumference of the chest to the height (C/H) and compares it with that of children of average size and the corresponding age. In 86 per cent of the boys the ratio corresponds to the normal; in 2.9 per cent it is higher, and in 13.5 per cent lower than the normal; 48.2 per cent of the girls had a normal ratio; in 51.7 per cent it was below, and in none above normal. When the C/H ratio was compared with that for children of the same height but older, it was found that 58.2 per cent of the boys were within the normal variations, 2.9 per cent had a higher, and 38.8 per cent a lower ratio; 37.5 per cent of the girls were within the normal range, 62.5 per cent had a lower, and none a higher ratio. Among children under 4 years of age the ratio was found to be normal in all the girls and in 83 per cent of the boys.

KAJDI, Baltimore.

INTERNATIONAL CONFERENCE ON AFRICAN CHILDREN, 1931. V. DE BUNSEN, Rev. internat. de l'enf. **11**:3 (Jan.) 1931.

The plan for a conference in 1931, devoted to the specific interests of African children, originated in 1928 with the late Eglantyne Jebb, founder of the Save the Children Fund. Miss Jebb's valiant spirit which has aroused so many European countries to the pressing needs of the children, following the World War, is to be carried on by her successors until all children are included in a movement that aims to atone in part for the suffering and neglect engendered not alone by the war but by centuries of ignorance and indifference.

The needs of the African children are to be considered under two heads: infant mortality and education. The conference proposes to make a survey of the conditions surrounding child life on this continent, to emphasize the positive work already done and to draw up a constructive program for the future. All European nations having interests in Africa are cooperating in the movement. The traffic in slaves, drugs, liquor and arms will receive particular attention; land policy, regulation of labor and taxation will be discussed.

AMESSE, Denver.

SUPERVISION OF HEALTH IN THE YOUNG. C. SCHIÖTZ, Rev. internat. de l'enf. **11**:22 (Jan.) 1931.

Child welfare has not provided adequately for two important periods: the preschool age and adolescence. In most countries infant welfare is well organized; in Norway, for example, the mortality for this age has decreased from 90 per thousand in 1900 to 49 in 1928. In Oslo, home of the author, it has dropped from 182 to 42.7. For children of school age, hygienic control has been rewarded by a general increase in weight and a great diminution in the number of cases of verminosis. After the elementary school period, however, young workers in the various trades are not well protected. Schiötz makes a plea for periodical medical examinations among adolescents and for supervision of their environment.

AMESSE, Denver.

Society Transactions

AMERICAN PEDIATRIC SOCIETY

Forty-Third Annual Meeting, Edgewater Park, Miss., April 15-17, 1931

PHILIP VAN INGEN, M.D., *President, in the Chair*

First Session

Wednesday Morning, April 15

PRESIDENTIAL ADDRESS: WHITHER ARE WE BOUND?

DR. PHILIP VAN INGEN, NEW YORK.

This article was published in full in the July, 1931, issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN, page 1.

FETAL PERITONITIS. DR. ISAAC A. ABT, CHICAGO.

Inflammatory processes in various organs occur during fetal life due to bacterial or nonbacterial agents. The escape of intestinal contents into the peritoneal cavity is one of the frequent causes of fetal peritonitis. The escape of meconium from the intestinal lumen is the most frequent cause of noninfectious fetal peritonitis. A favorite site for these perforations is the lower segment of the ileum. It is believed that before birth the meconium is free from bacteria. Shortly after birth, micro-organisms invade the intestinal contents, and, consequently, the meconium that escapes into the peritoneal cavity produces septic peritonitis.

In rare cases of malformation of the urinary tract, urine may gain access to the peritoneal cavity and in this way produce peritoneal irritation and inflammatory adhesions between the viscera.

While we are accustomed to think of appendicitis as commonly occurring during early infancy and childhood, we are surprised to learn of its occurrence during prenatal life. There are a number of well authenticated cases on record. In addition to the cases reported by Hill and Mason and by F. W. Jackson, the author presented the record of an infant who died at the Chicago-Lying-In Hospital on the third day after delivery. At autopsy a perforation was found near the apex of the appendix, which was covered with greenish, odorless, fecal material.

There is diversity of opinion concerning the significance and origin of peritoneal bands. It is well known from embryology that after the rotation of the large intestine, secondary attachments are formed which do not depend on previous inflammation. Occurrences of this kind may also play a part in the formation of the adhesions and bands that are found in the lower part of the abdomen and are associated with congenital defects of the female genitalia. It must be conceded that peritoneal bands and adhesions may be of developmental origin and may cause malformations that lead to perforations; however, there are numerous reports of peritonitis following perforation of the intestines, and it seems certain that fetal peritonitis is responsible for the adhesions that occur in these cases. The difficulty that arises is to decide whether the intestinal malformations that are frequently found are secondary to peritoneal inflammation and adhesions or whether they are caused by embryonal bands with the production of atresia, volvulus, secondary softening and perforation of the intestine.

Dr. Abt presented a detailed review of the literature and a description of several personally observed cases, including a discussion of the pathogenesis of fetal peritonitis and some of the controversial aspects of the subject.

DISCUSSION

DR. T. WINGATE TODD, Cleveland: I am glad to have had the opportunity of hearing Dr. Abt's presentation. Though not an embryologist, I have understood that, in a general way, the individual repeats his ancestral history in his development. We have recently had the opportunity to dissect forty monkeys and apes, including anthropoids, in order to learn the progressive or phylogenetic development of the intestine, large and small. So far as comparative anatomy is concerned, there is no evidence that justifies the assumption that peritoneal bands are a necessary part of the developmental progress.

I do not say that the comparative evidence absolutely contraindicates the embryonic development of such bands, but it does suggest that their presence implies something other than the factors ordinarily at work in guiding embryonic development. It is these extraordinary factors that Dr. Abt has presented for our attention.

PHYSICAL ANALYSIS IN THE ADOLESCENT PROBLEM. DR. T. WINGATE TODD, Cleveland.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

DISCUSSION

DR. HUGH WILSON JOSEPHS, Baltimore: Is it not possible that in dealing with high school boys and girls, one must consider that the majority of boys go to high school and college, whereas only certain girls go?

DR. T. WINGATE TODD: I was not speaking of particular students. I was speaking of the ordinary junior and senior high school student. We find the same thing in junior and senior high schools.

DR. HUGH WILSON JOSEPHS: Would not it apply, that most of the boys go to high school, but only certain girls do?

DR. T. WINGATE TODD: The superintendent told me that from 40,000 records made recently, he found that the boys who failed in promotion outnumbered the girls by more than 2:1. Most of this majority occurred in the junior high school period, during the ages of 12, 13 or 14, which seems hardly in line with that suggestion.

DR. BORDEN S. VEEDER, St. Louis: Many physicians have been considering the adolescent problem in their own practice, and the great physical problem, which I think we all realize, is the difference between the chronological and the developmental age. Nearly all adolescent activities, such as school work, have been based on a chronological age. I think that we are beginning to realize that many of our failures come from putting too much stress on a child of a certain chronological age during the adolescent period, when retardation of development exists.

I am very much interested in the subject, and I hope that a method can be worked out by which we can classify adolescent children for purposes of their school instruction, exercise, etc., on the basis of developmental age and not the chronological age.

DR. H. M. KNOX, JR., Baltimore: Did I understand Dr. Todd to say that there was much more rapidity of development from the age of 6 years on than between the ages of 3 and 6? I have always been under the impression that the child accumulated more new facts from the age of 3 to 7 than in any later period of the same duration.

DR. T. WINGATE TODD: Speaking in chronological terms, growth of the anthropoid brain, such as it is, is much more rapid than that of the brain of man. At or about the age of 6 years, the type of mental development in man undergoes a profound change in tempo. Until that date, there is a gross increase in the substance of the brain, with a rapid mental development. After that date, growth of the brain is slight, and mental development consists not in the introduction of wholly new phases but in the training of a mind, substantially complete in capacity, to finer discrimination by experience.

THE CHARACTER OF THE ELECTROLYTE LOSS IN DIARRHEAL DISEASE. DR. JAMES L. GAMBLE, Brookline, Mass., DR. ALLAN M. BUTLER, JR. (by invitation) and DR. CHARLES F. MCKHANN (by invitation), Boston.

The data presented were measurements of the excretion of sodium and potassium in the urine and stools of two infants suffering from diarrheal disease with vomiting. The specimens were collected during the first twenty-four hours after admission to the hospital. In both instances, so far as could be determined, no food or fluid had been retained for several days previously. The substances measured were, therefore, regarded as lost from the body. The finding considered significant was a loss of potassium which, relative to the loss of sodium, was much greater than could be explained on the usual assumption that the body fluid lost in diarrheal disease is entirely extracellular. It was also shown that the loss of potassium was greater than could be explained as the result of destruction of protoplasm due to fasting as estimated from the accompanying excretion of nitrogen. The loss of potassium above this value was taken to represent a reduction in the volume of intracellular fluid in the body. Published data indicating a similar event in ordinary fasting were cited. The results of the study are interpreted as supporting the use of potassium, as well as of sodium, in solutions designed to repair the effects of dehydration. The authors feel, however, that, if the solution is to be given intravenously, it would probably be unsafe to use a concentration of potassium much above the small value found in extracellular fluids.

HYPERPARATHYROIDISM AND SIMILAR DISEASES OF BONE. DR. THOMAS B. COOLEY, Detroit.

Two cases of extensive deforming disease of the bone originating in childhood were reported.

The first patient, a girl, aged 14, began having pain in the legs at the age of 10½ years. At 11½ years, she began to limp and later fractured the femur by slight trauma; she received prolonged treatment for nonunion. Extensive deformities of the legs, arms, pelvis and spine soon developed, and she lost more than a foot in height from scoliosis. Dental caries was extensive. Menstruation, which began at the age of 12, soon ceased. She lost all her hair. When admitted to the hospital, she had extensive demineralization and the formation of cysts in most of the bones. There were a wedge-shaped pelvis, extreme scoliosis, bowing of the arms and legs and thickening and porosity of the skull. Muscle tonus was poor. There was a palpable nodule in the lower pole of the enlarged thyroid gland. Clinical analysis of the blood showed: calcium, from 13.6 to 15.2 mg.; phosphorus, from 2.6 to 3.04 mg.; phosphatase, 0.251 mg., which is low for this type of disease. Urinalysis showed: calcium, 0.2751 and 0.3586 mg.; specific gravity, fixed. The calcium balance was not determined. The basal metabolic rate was + 23. The diagnosis was osteitis fibrosa cystica due to hyperparathyroidism.

On Dec. 6, 1930, the cystic parathyroid gland and a small amount of colloid goiter were removed. Slight tetany followed the operation, and was controlled by the application of calcium gluconate and parathormone. The blood calcium and phosphorus fell to 6.9 and 2 mg., respectively, and later rose to 10.8 and 4 mg., respectively. The calcium in the urine fell to 0.1111 mg.; fixation of the specific gravity disappeared, and the phosphatase was unchanged.

The improvement after the operation was all that could be expected, considering the deformity. There was marked euphoria and no pain. The menses returned; the patient gained 10 pounds (4,535.9 Gm.); the muscle tonus was good, and the color was better. Evidence of remineralization of the bone so early is doubtful.

The second patient, a girl, aged 11½ years, was seen in May, 1924. A year previously her hand had been caught in a door, and there was swelling of the fingers and the back of the hand. A little later she began to limp, because of softening of the head of one femur with upward displacement. Roentgenograms showed extensive, uneven demineralization, most marked in the pelvis, the left hand and the left femur; the skull was spared. Various diagnoses were made from

sections from the right ilium. Dr. Cooley's was enchondromatosis. At the Bone Sarcoma Registry, opinions concerning the diagnosis ranged from enchondromatosis to osteitis fibrosa. Later in the year, the progress of the disease led to the institution of roentgen treatment, with questionable benefit. She was seen again in the winter of 1930. She had not grown, and a goiter and tachycardia had developed. She was greatly crippled, and the lesions of the bone had progressed, with the formation of more cysts. Extensive metabolic studies were not possible. The blood calcium was 11.1 mg.; the phosphorus, 3.5 mg., and the phosphatase, 0.29 mg. The urine was normal. The basal metabolic rate was normal. The pulse rate was 110. It was thought that the changes closely resembled those of osteitis fibrosa, and that the cartilaginous growth might represent the osteoid tissue usually found.

Exploratory operation seemed justified, and was performed on Dec. 11, 1930. Half of an adenomatous goiter and at least one normal parathyroid gland were removed. There was no postoperative tetany. At the time of presentation, the patient was on a diet containing ample calcium and phosphorus, with viosterol. She had improved remarkably. She walked quite well, and had even danced. The swelling of the hand was lessening. Her color and the texture of her hair showed striking change. There was no evidence of new deposits of lime.

Slides from the two cases were shown.

The first case seems clearly to have been one of osteitis fibrosa cystica due to hyperparathyroidism. In the second case, there was no demonstrated hyperparathyroidism, and the surprising improvement is unexplained, though a psychic element is probable.

DISCUSSION

DR. JULIAN D. BOYD, Iowa City: Two years ago we made metabolic studies on a man who had hyperparathyroidism and resultant diffuse osteolysis. A tumor of the parathyroid gland was removed, and subsequently the evidences of disturbed metabolism subsided. Within three months there was evidence of good formation of bone, and now his skeleton has regained its normal density. In this patient, the syndrome was so well defined that we were led to expect that the clinical picture would be equally constant in other patients with similar conditions. Since that time, studies have been made of several other patients with dystrophies of the bone associated with metabolic disturbances, and the divergence of the findings has led to a confusion rather than a clarification of the issue.

One patient, a woman, aged 49, had an elevated serum calcium and a lowered serum phosphorus; she excreted large amounts of urine containing excessive quantities of calcium. She had no demonstrable generalized rarefaction of the bone. A diagnosis of hyperparathyroidism was made. A tumor of the neck was removed, which was found to be a thyroid adenoma, without demonstrable parathyroid tissue. No change in the mineral metabolism was noted subsequent to removal of the tumor.

Another patient, a woman, aged 69, presented the clinical and metabolic picture seen in the first patient. An adenoma of the thyroid gland was removed, and again in this instance parathyroid tissue could not be identified. Postoperative tetany developed, and after recovery from the operation, the metabolic anomaly apparently had been corrected.

Two girls, aged 14 years, had clinical evidences of localized osteitis fibrosa cystica. Metabolically, the two conditions were dissimilar, the excretion of urinary calcium being notably increased in one patient and definitely diminished in the other.

I wish to mention another type of dystrophy of the bone associated with evidences of metabolic disturbances; a group of children had roentgenographic evidences of active rickets, associated with low serum phosphorus and a diminished urinary excretion of calcium. The rickets was healed clinically by vigorous antirachitic treatment, yet the serum phosphorus remained persistently low. We have not been able to determine the cause or to correct the hypophosphatemia.

It is apparent that dystrophies of the bone may result from a variety of metabolic disturbances that produce similar clinical pictures. Individual metabolic study seems necessary in order to determine the most suitable therapeutic measures.

DR. T. B. COOLEY: Dr. Boyd's discussion emphasizes the apparent participation of the thyroid gland in some of these disturbances. There were tumors of the thyroid gland in both of the cases I reported. The proportion of the cases reported in which there were tumors of both the thyroid and the parathyroid gland is striking. One hesitates to talk vaguely of "polyglandular syndromes," but there seems to be reason to think of a probable involvement, primary or secondary, of more than one of the endocrine glands in some of these conditions. I recently saw an autopsy in a case of "renal rickets" in which hyperplasia of the adrenal cortex and the parathyroid glands and a remarkable eosinophilia of the pituitary body were found.

The findings of the Boston group in true osteomalacia seem to indicate a similarity to rickets rather than to hyperparathyroidism.

Second Session

Afternoon, April 15

ALKALI THERAPY. DR. ALEXIS F. HARTMANN and DR. MILTON J. SENN (by invitation), St. Louis.

In continuation of previous studies to determine when, in what manner and in what quantity alkali should be used in the treatment for severe acidosis, we have investigated the metabolism of sodium lactate in normal persons and in patients with various types of acidosis, with the following results:

In the normal subject, the intravenous injection of from 5 to 7 cc. of racemic sodium lactate per kilogram of body weight is followed by a return of the lactic acid content of the blood to its normal level in from one to two hours, during which time dextrose increases from 10 to 20 mg. per hundred cubic centimeters; the base-bicarbonate increases, on an average, 15 per cent by volume, and the inorganic phosphorus falls approximately 2 mg. per hundred cubic centimeters. Very little sodium lactate is excreted in the urine, but instead large quantities of base-bicarbonate (from 300 to 500 per cent by volume).

In all types of acidosis encountered, a similar metabolism of sodium lactate occurs, with, however, retention of base-bicarbonate in the body fluids until a normal level of total fixed base in the plasma has been reestablished. In the presence of marked damage to the liver and persistent anoxemia, the metabolism of sodium lactate is delayed. In the presence of marked renal insufficiency and a diminished electrolyte content of the body fluids, the base-bicarbonate resulting from the metabolism of sodium lactate may be retained, and alkalosis may result. If, however, a proper amount of the salt is given or if the normal electrolyte level of the body fluids is restored by a previous or simultaneous administration of Ringer's solution in adequate amount, such alkalosis may be easily avoided.

A satisfactory method of calculating the amount of sodium lactate to raise any observed plasma carbon dioxide content to approximately normal has been established as follows:

1. Body water (blood, lymph, spinal fluid, muscle water, etc.) equals from 60 to 70 per cent of body weight.

2. The $BHCO_3$ of the water of the entire body approximates the $BHCO_3$ of plasma.

3. One cubic centimeter of molecular sodium bicarbonate per liter of body water equals 2.24 cc. of carbon dioxide per hundred cubic centimeters (2.24 per cent by volume of carbon dioxide).

4. Therefore, the expected increase in per cent by volume of carbon dioxide equals $1 \times 2.24 \times \frac{\text{cc. of molecular sodium bicarbonate or sodium lactate}}{\text{liters of body water}}$.

5. The dosage of molecular sodium bicarbonate or sodium lactate to increase the carbon dioxide content of the body water to 60 per cent by volume equals $\frac{60\text{—carbon dioxide content}}{2.24} \times$ liters of body water, or (60 — carbon dioxide content) $\times$ (0.3 $\times$ body weight in kilograms).

In cases of nephritic acidosis the expected increase in carbon dioxide content a few hours after the administration of sodium lactate agrees very well with the foregoing values. In a case of severe acidosis associated with diarrhea, dehydration and oliguria, the observed increase is always somewhat less (average, 12 per cent by volume) than that expected, while in a diabetic case a difference either way may be noted, depending on the degree of acidosis, the lowering of the hydrogen ion concentration and the extent of oxidation of the organic acid.

Because of the gradual conversion of sodium lactate into alkali, which permits the organism to maintain a normal $BHCO_3 = H_2CO_3$ ratio, because of its stability, ease of preparation, sterilization and compatibility with Ringer's solution and dextrose and because it may be administered intravenously, subcutaneously, intraperitoneally or by mouth (in the absence of vomiting and diarrhea), it is felt that sodium lactate is, in general, much superior to sodium bicarbonate.

DISCUSSION

Dr. Bengt K. Hamilton, Chicago: I was much impressed with how well Dr. Hartmann has succeeded in restoring the lost base in the bodies of dehydrated infants, but I wonder whether or not the method of calculation was unnecessarily refined. My question is whether by calculating the dose simply by taking so many cubic centimeters of the solution per kilogram of body weight, Dr. Hartmann would not have found just about the same amount of variation as by this very refined method of calculation?

Dr. S. W. Clausen, Rochester, N. Y.: I should like to know whether Dr. Hartmann uses racemic lactic acid or dextrolactic acid. It is rather surprising to learn that the lactic acid kept in the laboratory acts as well as that found in the body.

Dr. A. F. Hartmann: In answer to Dr. Hamilton's question, I think that our method of calculation not only is not too refined, but is actually crude. I feel certain that the dosage of alkali given will determine the resultant carbon dioxide content of the blood to a considerable extent, and in some of our earlier studies we have given rather small amounts of both sodium bicarbonate and sodium lactate, with only a small resulting increase in the carbon dioxide content of the plasma. What rise occurred, however, agreed well with what might have been predicted by our present method of calculation. I am sure, however, that if excessive doses of alkali are given, the observed carbon dioxide contents will frequently not be abnormally high, provided a determination is not made within a few hours after the administration of the alkali. This would mean, however, only that renal activity was sufficient to excrete rapidly any excess of alkali given.

In answer to Dr. Clausen's question, I might say that we have not yet investigated the effect of dextrolactic acid. From the work of Riegel and Abramson and that of Eggleton and from our own results, it seems fairly clear, however, that racemic lactic acid is utilized almost completely. We are planning to continue our studies on this point, and will study the effects of dextrolactic acid.

Hypoglycemia in Infants: A Preliminary Report on Babies with Symptoms Referable to the Autonomic Nervous System. Dr. C. A. Aldrich and Dr. G. F. Munns (by invitation), Winnetka, Ill.

Such alarming and disconcerting symptoms as cyanosis, choking, dyspnea, stridor, asthma, projectile vomiting and sudden death in young infants are often inadequately explained either by clinical observations or by observations found at

autopsy. It seems probable that they may be due to what may be called, for want of a better term, functional disturbances, possibly of the autonomic nervous system.

Insufficiency of the suprarenal medulla, an integral part of this system, might be expected to cause a reduction in the level of the blood sugar in addition to such symptoms, since epinephrine tends to elevate this reading. It is conceivable that the amount of sugar in the blood may be a fair index of the type of autonomic activity in the patient as well as of the relative functional activity of the suprarenal medulla and the islets of Langerhans. The fact that the symptoms encountered are often found at a time when the suprarenals are undergoing marked involution, soon after birth, may be significant. McLean and Sullivan reported low figures for blood sugar in three patients with "status thymo-lymphaticus" shortly before death, and pointed out that this could be explained on the basis of hyposuprarenalism.

Before feedings, samples of blood were taken from the heels of babies with symptoms, and control examinations were made on specimens from normal babies of approximately the same age. The clinical material was furnished by the service for the newly born at the Evanston Hospital, by private practice and by the helpful cooperation of pediatricians who were attending patients in this hospital. All specimens were examined by Miss Iva Palmer in the pathologic department, the Byrd modification of the Folin-Wu micromethod, which requires only a few drops of blood, being used.

Estimations during fasting of the blood sugar of nineteen infants with symptoms averaged 0.06 per cent; the highest was 0.078 per cent, and the lowest, 0.025 per cent. For nineteen controls of the same age, the average reading was 0.084 per cent; the highest was 0.111 per cent, and the lowest, 0.070 per cent. The marked difference in the readings for the infants used as controls and for those of the patients, 0.024 per cent, prompted this report. The patients all showed definite hypoglycemia, while the figures for the infants used as controls compared favorably with those of other observers.

Three possible explanations for these readings confront us: (1) that the hypoglycemia is due to hyposuprarenalism, (2) that it is due to hyperinsulinism and (3) that it results from lack of glycogen in the liver or from hepatic dysfunction. It is considered that both the hypoglycemia and the symptoms can be explained on the basis either of suprarenal insufficiency with resulting decreased sympathetic activity or of vagotonia. Whether they actually are due to one or both of these conditions or to the hypoglycemia itself is an open question. The results are in keeping with, but not proof of, the theory that suprarenal insufficiency is the primary cause of the symptoms.

DISCUSSION

DR. HENRY J. GERSTENBERGER, Cleveland: I would like to ask Dr. Aldrich whether he made dextrose tolerance tests on the infants; that is, whether he gave them a definite amount of dextrose and then observed the blood sugar level at fifteen or thirty minute intervals. If he did so, what were the results?

DR. SAMUEL Z. LEVINE, Flushing, N. Y.: I would like to know whether you can attach any importance to the reduced glycogen reserve in the liver of the new-born infant as a possible contributory cause of hypoglycemia.

Normally, in the new-born infants the respiratory quotient falls within the first two or three days, indicating the utilization of fat and protein only at the end of that time. In infants who vomit or have cyanotic attacks, the possibility is that they do not eat as well as the normal newly born infant. Because of the lack of ingestion of carbohydrates, the glycogen reserve in the liver would be depleted more rapidly than in the normal infant. This may contribute to a low sugar content of the blood in some newly born infants with symptoms, without necessitating hyposuprarenalism.

DR. A. F. HARTMANN, St. Louis: The paper presented by Dr. Aldrich was interesting, especially in the light of the values for blood sugar we obtained in new-born infants. We used the zinc sulphate precipitation method of Somogyi

to get "true" values for dextrose, and found, in line with Dr. Levine's suggestion, that during the first four days the true blood sugar content falls steadily. In normal infants, we noted levels of blood sugar as low as 40 mg. per hundred cubic centimeters. We also determined the amount of sugar in the blood before and after feedings. In infants who had not previously been getting adequate amounts of food, very little rise occurred after feeding. In those in whom there was an apparently good reserve of glycogen, a greater rise in blood sugar occurred.

DR. H. W. JOSEPHS, Baltimore: I was interested in the case of jaundice. Several years ago I had occasion to study a case of cirrhosis of the liver with jaundice. During the time when there was an exacerbation of the jaundice, the child had a tendency toward hypoglycemia after short periods of fasting, whereas about four hours after a meal there were no symptoms of hypoglycemia. The blood sugar fell more rapidly than in the normal child.

Studied later, when the jaundice was less marked, the child did not show the marked drop in blood sugar.

I would like to ask Dr. Aldrich whether determinations of acetone in the body were made, and also whether there was a possibility that in the cases in which vomiting occurred low blood sugar was noted during fasting.

DR. C. A. ALDRICH: I purposely tried not to create the impression that we thought the question of the cause of hypoglycemia was settled. I was particularly anxious merely to show that it existed in infants of this type, and to give two or three possible explanations for its presence. The older infants who had hypoglycemia with asthma were in a good nutritional state.

We showed the results of experiments on nineteen infants used as controls and on nineteen patients; as far as we could tell, the infants used as controls were normal and of the same age. With the method used, the infants used as controls had a blood sugar of 0.025 per cent higher than those who presented symptoms. We have not made any dextrose tolerance tests in these cases. A great deal of work has been done along that line, showing the glycemic curve following feedings in infants.

One investigator showed curves differing in premature infants, in infants in good nutritional state and in those with various nutritional conditions. The curves also vary with various kinds of feedings; for instance, with protein milk. Specimens were taken from most of the infants with symptoms early in life, a great many on the first or the second day of life. It seems to me that they were taken early enough and that we had enough infants of the same age used as controls, so that we probably were not dealing with malnutrition or with a depletion of dextrose in the liver. Had the hypoglycemia been due to the starvation of the first few days, our infants used as controls also should have had low figures.

In the cases in which vomiting occurred, there is a possibility that the hypoglycemia was due to the loss of food. However, because of the treatment that was being instituted, several of the infants with low blood sugar actually gained in spite of the vomiting. We gave them enough food to make them gain, so I doubt if the hypoglycemia was caused by starvation.

There is a great deal of interesting literature on the normal infant's blood sugar. I have not seen any figures as low as those Dr. Hartmann quoted, but I assume that the difference is due to technic. The lowest figure I have seen that was considered as normal is 0.070 per cent. Average for newly born infants range around 0.085 per cent.

We have not done any work on the acetone bodies.

A CLINICAL METHOD FOR THE STUDY OF THE PROTEOLYTIC LEUKOCYTIC ENZYME IN LEUKEMIA. DR. JEAN V. COOKE, St. Louis.

Previous methods used in the study of the proteolytic enzyme in the blood have usually required considerable amounts of blood and have been only roughly

quantitative. It is agreed that the granular polymorphonuclear leukocytes and myelocytes possess a high degree of proteolytic activity. The presence of such activity in the nongranular cells found in cases of acute leukemia, however, has been a matter of dispute. One point of interest concerns the possible relation of these cells to normal lymphocytes, which have very little proteolytic activity, or to myeloblasts, which presumably are similar to the myelocytes in this respect. Some observers have found these cells rich in proteolytic activity, while others have found no such activity.

The method used in this study differs from the methods previously employed in that the hemoglobin of the blood is used as a substrate on which the leukocytic protease may act, and in that the use of microchemical methods allows determinations to be made on 10 cc. or less of blood. This procedure permits the study of the same patient at different times, and by the recording of the results in milligrams per hundred cubic centimeters of digested nitrogen, offers a means of quantitative comparison between different samples.

At approximately the same time that a total and stained differential leukocyte count is made, a sample of venous blood is placed in a test tube with a small amount of powdered sodium oxalate to prevent clotting. After the total volume has been marked on the tube, the specimen is centrifugated and the supernatant plasma removed and discarded, care being used to avoid taking away any cells. The cells are then washed three times in 0.85 per cent salt solution to get rid of the last traces of serum, and after the last washing and removal of the supernatant fluid, distilled water is added to the tube until the original volume is reached. This is well shaken to distribute the leukocytes evenly through the hemoglobin, and samples of 3 cc. are placed in large test tubes with a volumetric pipet. These are treated as follows:

1. Control tube. To this tube are added 24 cc. of distilled water, 3 cc. of 10 per cent sodium tungstate and 3 cc. of two-thirds molar sulphuric acid. After shaking and filtering, the nonprotein nitrogen in a sample of the filtrate is determined, and the milligrams of nonprotein nitrogen per hundred cubic centimeters are calculated.

2. Protease in neutral medium. After the addition of 7 cc. of water and a small quantity of toluene, the tube is incubated at 37 C. for five days. Then 3 cc. of 10 per cent sodium tungstate, 3 cc. of two-thirds molar sulphuric acid and 14 cc. of water are added, and the determination of nonprotein nitrogen in the filtrate is made.

The procedure outlined is the well known method of Folin and Wu, and the determinations of nonprotein nitrogen were carried out by acid digestion and nesslerization, as usual. The results are expressed as milligrams of nitrogen per hundred cubic centimeters of blood.

With this method normal blood shows a protease figure that varies from 100 to 125 mg. per hundred cubic centimeters of nonprotein nitrogen; five patients with chronic myeloid leukemia and counts varying from 15 to 300,000 showed figures of from 250 to 1,680 mg. per hundred cubic centimeters of nonprotein nitrogen.

Twenty estimations at various times on thirteen patients with acute leukemia showed figures varying from 17 to 750 mg. per hundred cubic centimeters of nonprotein nitrogen. In general, the higher readings were found some months before death, when the patient's general condition was good, and there was a definite tendency for the leukocytic protease to fall to a low figure in the terminal stage of the disease.

THE RELATIONSHIP OF INTESTINAL SYMPTOMS TO ENTERAL AND PARENTERAL INFECTION. DR. MCKIM MARRIOTT.

This paper was read by title.

CAROTINEMIA AND RESISTANCE TO INFECTION. DR. S. W. CLAUSEN, Rochester, N. Y.

A statistical analysis of respiratory infection occurring in a group of 700 children under 3 years of age and a consideration of their diets showed that a relative deficiency of vitamin A may exist and be correlated with the severity of the infection. Since carotin has recently been shown to be a percussor of vitamin A, it seemed important to study the relationship between carotin in the organism and infection. Since chemical methods exist for its determination in blood and tissues, it became possible to study by direct methods the behavior of carotin in infections. We have shown that carotin is present in the blood at birth, that it rises by the age of 2 years to an average value of from 0.075 to 0.100 mg. per hundred cubic centimeters in the plasma by the end of the second year, and that the average values in healthy children are low in December, January and February. Starvation causes a slow disappearance of carotin from the blood, but severe infection causes its rapid disappearance. In a small series of cases of severe otitis media, rather rapid improvement seemed to follow the elevation of carotin in the blood; however, this did not invariably occur. In infants between the ages of 6 and 24 months, the greatest number of severe infections were found in those who had not received cod liver oil or vegetables and whose plasma carotin was extremely low. The plasma carotin of infants who had received adequate amounts of cod liver oil was nearly the same, whether or not mild or severe infections were present. In a series of cases of moderately severe scarlet fever, plasma carotin was determined within three days of the onset; the group of cases in which complications did not subsequently occur had nearly the average normal carotin; the group in which severe complications subsequently developed had, at the onset, very low plasma carotin. It is believed that these observations indicate that in children carotin may act as provitamin A, and that in the absence of sufficient vitamin A, carotin may confer some degree of protection against the development of severe complications in the common respiratory infections of infancy, and also in such a disease of later childhood as scarlet fever.

DISCUSSION

DR. I. MCQUARRIE, Minneapolis: It has been empirically observed that, on the average, diabetic patients have fewer infections of the upper respiratory tract than other persons. I was impressed with that fact myself while I was in a large diabetic clinic for a year or two, and I wonder whether carotin might not be the factor. Such patients have a higher degree of carotinemia than any other group because they are given so much carotin in the form of green vegetables.

We have previously attributed this to the increased proportion of fat in the diet or to the mild degree of dehydration of the tissues that is frequently present.

DR. H. J. GERSTENBERGER, Cleveland: I wonder whether the carotin that Dr. Clausen finds plays a beneficial rôle in protecting infants and children against infections. Does it not produce this good result over another route than that of increasing the fat-soluble A content of the body?

Infants and children who receive liberal amounts of cod liver oil do get infections, and I doubt whether it is feasible or possible to put as much of the fat-soluble A factor into the body by feeding substances rich in carotin as one can by giving cod liver oil. However, I have felt for some time that pigments may play an important rôle in nutrition.

DR. O. M. SCHLOSS, New York: I have been much interested in Dr. Clausen's results, which he has presented in a very conservative way. It would be of considerable interest to feed carotin to children subject to infections of the upper respiratory tract and to determine the carotin in the blood in susceptible children at various times to see if the quantity of carotin is smaller during the course of infection than during periods of immunity. It would also be of interest to know if the amount of carotin were smaller in children who were susceptible to infectious colds than in those who were relatively immune.

DR. S. W. CLAUSEN: We have not made extensive feeding experiments on children without infections, but we are accumulating data on children who come to the hospital with otitis media. These data are not easy to evaluate because of the great variability in the course of that disease, but I have a strong impression that cases that seemed to be very severe and we thought at first were destined to develop into mastoiditis not infrequently pursued a very mild course when it was possible to raise the carotin in the blood by the feeding of carrots (an ounce a day to an infant, and an ounce or an ounce and one-half twice a day to an older child). This is not an invariable effect, and we have observed one or two children with a fairly high amount of carotin in the blood in whom infections developed.

We have also observed what Dr. McQuarrie pointed out, that diabetic and epileptic patients receiving a diet high in carotin are less likely to be attacked by severe infections; nevertheless, this year we have observed a few diabetic patients with scarlet fever. At the onset, the carotin in the blood was fairly high, but it rapidly became lower, probably as the result of infection and of a change in the diet. Some of the patients had mastoiditis as a final complication of scarlet fever.

I do not believe that we should expect too much protection from carotin. It is only one of the many nonspecific factors that may play a rôle in preventing serious infections.

In answer to Dr. Gerstenberger's comments, I wish to say that cod liver oil does not prevent serious respiratory infections in isolated cases. If one studied a large enough series of cases, perhaps several hundred, I am convinced that one would get a definite correlation between the previous intake of cod liver oil and the course of infections that might develop in the infant. One can also show that rickets is not correlated with the presence or the absence of infections, which, I think, argues that vitamin A rather than vitamin D is the important factor in the nonspecific resistance conferred by cod liver oil.

THE OCCURRENCE AND SIGNIFICANCE OF THE ENTERAL ABSORPTION OF ANTIGENIC PROTEIN BY INFANTS. DR. ARTHUR F. ANDERSON (by invitation), DR. OSCAR M. SCHLOSS and DR. ROBERT O. DUBOIS (by invitation), New York.

The addition of a protein never before ingested to the diet of normal infants leads to the absorption of a small amount of the protein in antigenic form, as is evidenced by the appearance of specific precipitin in the blood and, in some instances, by the production of cutaneous hypersensitivity. Such absorption is of short duration, which is shown by the speedy disappearance of precipitin from the blood despite the continued ingestion of the protein. The ingestion of different varieties of protein successively provokes the same phenomenon.

The absorption of antigenic protein by normal infants is similar to that observed in those suffering from marasmus or diarrhea, but differs strikingly in certain respects. In normal infants less protein is absorbed, the absorption is of shorter duration, and the precipitin appears much earlier.

It would seem that there are three protective factors concerned in the enteral absorption of antigenic protein. The digestive enzymes tend to disrupt the protein molecule to nonantigenic derivatives. In addition, there is an unknown mechanism that renders the intestinal wall impermeable to antigenic protein. Despite these protective influences, under certain conditions a small amount of undigested protein enters the circulation. This provokes the production of precipitin, which probably represents a protective mechanism.

The possible clinical significance of the absorption of antigenic protein was discussed.

DISCUSSION

DR. J. D. TRASK, New Haven, Conn.: Did you apply your technic to children with eczema? After the precipitin had disappeared, did a substance that would inhibit the test with precipitin appear in the children's serum?

DR. T. M. RIVERS, New York: Were you certain that the antigen did not come in contact with the upper respiratory tract?

DR. I. MCQUARRIE, Minneapolis: Have you used bacterial protein? Since enteral vaccination against typhoid seems to be coming into vogue, I wondered whether this procedure might not serve as a means of testing the relationship of the reactions of precipitin to immunity.

DR. L. E. HOLT, Baltimore: Were the various milk proteins studied independently?

DR. O. M. SCHLOSS: In answer to Dr. Trask's question, we observed a number of cases of eczema, and at times the patients showed precipitin in the blood and at times they did not. It has been of interest, however, to observe a number of patients who were sensitive to milk and to egg; in these cases, the precipitin was apt to be absent or to be present during periods of improvement.

We have given considerable thought and attention to the possibility of inhibition of the precipitin test by antigen. For example, in what we have called the pre-precipitin stage of marasmus, a period in which there is no precipitin in the blood, it occurred to us that the negative reactions to precipitin tests were perhaps due to the fact that antigen was passing through the intestinal tract in sufficient quantity to combine with the precipitin and thereby mask its presence.

In order to obtain information on this question, seven infants were fed food free from milk protein for ten days or more. In no instance did a reaction to the test with precipitin that was previously negative become positive. This we felt demonstrated that the negative reactions to the precipitin tests were due to the absorption of precipitin.

In answer to Dr. River's question, we did not feel that in the ordinary course of the ingestion of food sufficient remained in the nasopharynx to permit its absorption. We merely attempted to give information on the intestinal absorption of antigenic protein when the food was given in the normal way. It would be possible, of course, to answer Dr. River's question definitely by carefully feeding a number of infants by stomach tube and determining whether sufficient antigen was absorbed to cause a positive reaction to precipitin.

Third Session

Thursday Morning, April 16

GLOMERULAR CHANGES FOLLOWING INJECTIONS OF STREPTOCOCCUS VIRIDANS. DR. HENRY F. HELMHOLZ, Rochester, Minn.

The study of experimental nephritis in the rabbit has been complicated by the frequency with which this animal shows spontaneous lesions of these organs. The earlier investigation almost without exception ascribed the lesions to the material injected. As has been shown most clearly by Bell and Hartzell, all of the lesions described are found spontaneously in the rabbit's kidney.

Bell, Clauson and Hartzell, Longscope, Birghaug and Howard and LeCount were unable to produce characteristic glomerular changes. In 1926, Duval and Hubbard reported the production of glomerulonephritis in rabbits following the injection of *Streptococcus hemolyticus* and its toxins, but the illustrations in their paper are not convincing; repetition of the work by Ruth, Warfield and Euzer gave negative results.

With the exception of Clawsen, who injected particulate material with *Streptococci viridans,* no one has stated that a large number of glomeruli were affected. Recently, Blackman, Brown and Rake reported the production of acute nephritis by means of a pneumococcus autolysate.

My experiments consisted of a series of three groups: In the first two groups, the rabbits were given injections of organisms, one with *Streptococcus hemolyticus* isolated from the middle ear of a patient in whom nephritis developed, and the other with *Streptococcus viridans* isolated from the urine of a patient with acute

hemorrhagic nephritis. The animals were given from one to nine intravenous injections. These experiments gave negative results. The third series consisted of seven animals that were given injections of streptococci isolated from patients with subacute bacterial endocarditis; four received the first injections subcutaneously in milk, and the next two intravenously; the other three received all of their injections intravenously.

Two animals died during the experiments. The remaining animals were killed on March 30 and 31, 1931. They had received their first injection on May 7, 1930. From rabbit D 777, the left kidney had been removed two weeks previously. On Jan. 29, 1931, a small piece of the pole of the right kidney was removed. On Jan. 16, 1931, the left kidney of rabbit D 823 was removed under aseptic conditions for histologic examination.

Rabbit D 823 showed the most marked changes in the left kidney, which was removed on January 16. Practically all of the glomeruli showed changes, as seen by the Mallory-Haidenhalm stain; with hematoxylin and eosin, only about 60 per cent showed the hyaline changes that may involve a loop or almost the entire glomerulus. In some glomeruli there was a definite increase in the number of cells, and in areas evidence of nuclear fragmentation was seen. Congo red stains for amyloid showed that the hyaline material took this stain. In the right kidney, which was removed on March 30, the changes were more intense; 96 per cent of the glomeruli showed changes in sections stained with hematoxylin and eosin. In spite of the marked involvement of practically all the glomeruli of one kidney, of marked sclerosis of one pole of the remaining kidney, of pyelitis caused by the colon bacillus complicating the final two months of life and of pregnancy, the blood urea was only 28. Six weeks previously, it had been 22.

In the small piece of cortex removed on January 29 from animal D 777, the changes were not so far advanced, showing more cellular reaction and adhesions between the tuft and the capsule, and only an occasional glomerulus had a hyaline area in it. Sections from rabbit D 777 stained with hematoxylin and eosin showed an involvement of slightly over 20 per cent. The most marked involvement showed a picture resembling the crescents of glomerulonephritis. The urea was never over 22 mg. per hundred cubic centimeters.

The other three animals received intravenous injections, the first on May 20, 1930. Animal D 949 died 62 days, animal 947, 165 days, and animal 845, 215 days after the first inoculation. Animal 845 showed the most marked changes. Practically all of the glomeruli showed marked degeneration, and few capillaries could be seen, most of which were a hyaline mass with a few nuclei and some fragmentation. Congo red stain showed that this material was amyloid. In rabbits D 777 and D 823, an abscess developed at the original site of inoculation; animal 845 showed no suppurative lesion at any time during life or at postmortem examination.

Four of the five rabbits available for study showed definite pathologic changes of the glomeruli, and two, involvement of practically all of the glomeruli. In view of the fact that widespread changes of this kind have not been observed to occur spontaneously, it seems fair to assume that these changes were produced by the repeated injection of the *Streptococcus viridans*.

COMPARATIVE IMMUNOLOGY IN PEDIATRICS. DR. JAMES D. TRASK, New Haven, Conn.

The great number of variations in age in infectious diseases are but partly understood. This is especially true of the variations that are not so much concerned with the mere incidence of infection as with the age incidence in the clinical features of disease. The only infectious disease in which the latter aspect has received much study is tuberculosis. In this the application of the test with tuberculin to large groups of persons has established the distribution of allergy to tuberculin. Besides this principle, there are probably others of an immunologic nature which, if understood, might explain some variations in the reaction to infection. If a science of comparative immunology existed, much light might be

shed on the mechanism of infectious disease. As such a science is lacking, the application of immunologic methods to the study of scarlet fever and pneumococcal pneumonia at different periods of life shows what might be the function of comparative immunology.

Cooke showed that new-born infants do not react to scarlet fever toxin even when neither they nor their mothers possess scarlet fever antitoxin. In other words, he showed that a child is not born with, but acquires during life, susceptibility to scarlet fever toxin. A question for further investigation then arises. Does this fact separate scarlet fever toxin from all other bacterial toxins, or are there other toxins to which man is born insusceptible, becomes susceptible and then immune?

It has been found that children recover from pneumococcal pneumonia more readily than adults, but not because of the possession of a better method of making type-specific antibodies, since it was determined that they do not make antibodies more quickly or in even as high a titer as do adults. Therefore, it is suggested that adults acquire a susceptibility not possessed by children to the products of the pneumococcus.

This paper is taken from a report, prepared by Dr. J. V. Cooke and Dr. J. D. Trask, for the White House Conference on Child Health and Protection.

DISCUSSION

DR. B. SCHICK, New York: As far as the reactivity of the skin of new-born infants is concerned, there is no doubt that the skin of these infants is much less reactive than the skin of other children. From my tests on diphtheria, I know that it is difficult to produce a positive reaction in new-born infants; higher concentration is necessary. At first I thought that every new-born infant would give a negative reaction, owing entirely to the fact that antibodies were transmitted, but later I realized that the sensitivity of their skin is much lower. Gröer studied the reactivity of the skin during the first days of life in comparison to that of other age periods. What was found in connection with the test for diphtheria was also found to be true of the test for tuberculosis. Higher concentration was again found to be necessary in order to produce a positive reaction.

THE CAUSE OF POLIOMYELITIS. DR. MERLIN L. COOPER (by invitation), Cincinnati.

Studies of the etiologic factor of poliomyelitis have revolved around the filtrable virus, the globoid body and the streptococcus. My investigations have been associated with the streptococcus.

Green-producing streptococci have been isolated from the spinal fluids of 65 per cent of the cases of poliomyelitis studied, including paralyzed and abortive ones. In the majority of instances, flaccid paralysis and death of the rabbits have occurred when either concentrated spinal fluid or cultures of it were injected intracerebrally.

Streptococci have been recovered from the brains of rabbits that were given injections, and they have then reproduced the same condition of paralysis and death in rabbits given subsequent injections.

The streptococci are easily visible in stained smears and in sections of the substance of the brain of these animals.

Human convalescent poliomyelitis serum afforded complete protection to rabbits when given subcutaneously one hour before, one hour after or intravenously one hour before the intracerebral injection of streptococci. The intravenous administration of convalescent serum one hour after injection did not give protection. In an unprotected rabbit employed as a control in this particular experiment, which was given an injection in a similar way of an equal amount of the same culture, flaccid paralysis developed, and the animal died in twenty-nine hours.

When the nostrils of rabbits were packed with sterile gauze saturated with a suspension of the streptococci, paralysis developed, and the animals died within from six to seven weeks. Streptococci were then recovered from the brains.

When the streptococci were injected intravenously rather than intracerebrally, paralysis and death occurred in from eight to nineteen days, and streptococci were recovered from the brain.

Blood cultures obtained shortly before the death of the rabbits, no matter how the injection was made, have always been negative. This is contrary to the experience after the injection of other types of streptococci.

Filtrates of these streptococci, after passing through the finest Berkefeld candles, have remained sterile to cultural methods. Rabbits have remained normal when this filtrate was injected intracerebrally, intravenously, submucously and intraperitoneally, and when used to saturate gauze for packing in the nostrils.

Paralysis has developed in rabbits and they have died after the injection of streptococci that had been isolated from a patient three months previously. These cultures had been transplanted every five days during the three months' period and had been cultivated aerobically on the surface of suitable mediums.

When specimens were taken from the throat for culture in an abortive case of poliomyelitis three days after the onset of symptoms, it was found that 75 per cent of the micro-organisms were green-producing streptococci of a type morphologically and culturally similar to those isolated from spinal fluids. This particular streptococcus produced flaccid paralysis and death in rabbits.

In some instances, similar streptococci from the throats of patients with late abortive poliomyelitis or of late convalescents with paralysis caused meningitis in rabbits; in others, they were without effect; however, the typical paralysis already described was not produced.

Other micro-organisms, including hemolytic streptococci and meningococci, when injected intracerebrally produced meningitis and death, but not paralysis.

Sterile saline solution and the substance of the brain of normal rabbits have been injected without producing symptoms.

Specimens from the brains of several normal rabbits, which were not given experimental injections, have been cultured in the same way as those from the animals described, and in all cases the cultures remained sterile.

Serologic experiments indicate that the blood serum of artificially immunized rabbits contains precipitins specific for similar streptococci from the spinal fluid of patients with poliomyelitis.

In an as yet incomplete pathologic study of sections of the brains and cords of a few paralyzed animals, it has been found that all have lesions suggestive of poliomyelitis; in one case that was studied more in detail, there was a marked perivascular infiltration in the brain and cord similar to the lesions found in poliomyelitis.

DISCUSSION

Dr. T. M. Rivers, New York: This paper is a continuation of investigations that have been going on for fifteen years, and such communications always lead to heated arguments.

I do not doubt that streptococci have been recovered from the spinal fluid of patients with poliomyelitis, nor do I doubt that streptococci have been recovered from the brains of persons who have died of poliomyelitis. I am surprised, however, that Dr. Cooper has obtained such a high percentage of positive cultures from the spinal fluid when other bacteriologists have failed.

I do not doubt that the streptococci with which Dr. Cooper is working when injected into rabbits in the manner indicated produce the effects illustrated on the screen. They undoubtedly cause paralysis.

In spite of all that I have admitted, I still doubt that proof has been brought to show that the streptococci isolated from patients and causing paralysis in rabbits represent the etiologic agent of poliomyelitis.

I would not repeat what has been said over and over during the last fifteen years were it not for the implications carried with the presentation of such a paper, viz., on the use of antistreptococcal serum in the treatment of children with poliomyelitis. It is all right to discuss such experiments here, but caution should be exercised in the use of the serum in clinical pediatrics.

I could talk for an hour or two about why I do not believe that the streptococcus is the cause of poliomyelitis; however, I shall only point out several potent reasons why it is not considered the etiologic agent.

It is generally accepted that monkeys are susceptible to the virus of poliomyelitis. When the monkey is used as a test animal, the virus of poliomyelitis is practically never demonstrable in the spinal fluid of patients. Dr. Cooper, however, produced paralysis in rabbits with spinal fluids and with organisms grown from the spinal fluid of patients.

When the virus of poliomyelitis is injected intravenously into monkeys, it rarely, if ever, produces paralysis. Dr. Cooper, however, was able to cause paralysis in rabbits by the intravenous inoculation of streptococci.

Reinoculation and neutralization are the best means of identifying poliomyelitis virus. Experiments in the production of protection are less reliable and may, at times, lead to confusion. Monkeys that have recovered from infections with the so-called poliomyelitis streptococcus are susceptible to the true virus of poliomyelitis, and vice versa. Antistreptococcal serums do not neutralize in vitro the true virus of poliomyelitis, while convalescent human serum does. In other words, there is no immunologic evidence of a relation between the two agents. Dr. Cooper said nothing in regard to reinoculation and neutralization. He did not, because, so far as I know, no one has shown that the rabbit is susceptible to the true virus of poliomyelitis, and such experiments must be conducted on animals susceptible to the disease.

Finally, the pathologic picture produced in rabbits by streptococci isolated from patients with poliomyelitis is not similar to that caused in monkeys and man by the true virus. Furthermore, the rabbit is a notoriously poor animal for the study of experimentally induced lesions in the nervous system, because it is subject to at least two diseases, virus III infections and infections with *Microsporidia*, which may spontaneously attack the brain and cord.

DR. M. L. COOPER: We entered on this study of poliomyelitis in order to find information regarding the presence or absence of micro-organisms in the spinal fluid. The results, which I have explained, and the moving pictures, which you have seen, have portrayed our accomplishments. These findings have aroused our interest and curiosity regarding the association of these micro-organisms with this disease. We feel that the frequency with which they have been found warrants further studies in which monkeys will be used for experiments on reinoculation, cross immunization and serum protection.

Our filtration experiments have given negative results. We have filtered cultures through the finest Berkefeld candles, and have injected the filtrate into animals intravenously, intracerebrally, submucously and intraperitoneally. Symptoms did not develop in any of these animals, and, as far as we could tell, all of them remained normal. The cultures used in these experiments had been isolated three months previously from the spinal fluid of patients with poliomyelitis.

TREATMENT FOR RESPIRATORY PARALYSIS IN POLIOMYELITIS WITH THE DRINKER RESPIRATOR. DR. JAMES L. WILSON (by invitation), Boston.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

DISCUSSION

DR. I. MCQUARRIE, Minneapolis: We have one of the Drinker respirators in Minneapolis and have had four occasions to use it. In one of the cases transient tetany developed apparently from forced overventilation of the lungs. The facial muscles were twitching when the physician entered the patient's room, but there was, of course, no carpopedal spasm because of the motor paralysis. When the apparatus was shut off for a time to allow a reaccumulation of carbon dioxide in the tissues, the spasm disappeared.

The apparatus is undoubtedly indispensable in certain cases, but it requires the constant attention of a specially trained personnel.

DR. J. L. WILSON: I do not wish to give the impression that the machine can be used carelessly by any one without danger of overventilation. The danger, however, does not appear to be great.

THE RELATIONSHIP BETWEEN THE SKIN AND THE BODY TEMPERATURE AND SOME PHYSIOLOGIC FACTORS INVOLVED. DR. FRITZ B. TALBOT.

The paper was read by title.

PULMONARY DISEASES: A STUDY OF FIVE THOUSAND EIGHT HUNDRED AND SIXTEEN CHILDREN, WITH SPECIAL REFERENCE TO THE CHILDHOOD TYPE OF PULMONARY TUBERCULOSIS. DR. CHESTER A. STEWART (by invitation), Minneapolis.

This paper will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

DISCUSSION

DR. H. F. HELMHOLZ, Rochester, Minn.: I have been extremely interested in some of the points Dr. Stewart brought out. I think that perhaps the most important one is that regarding the resistance of the infant and small child to tuberculosis.

I think it is generally stated that the small infant has no or only very slight resistance to tuberculosis, and that this is responsible for the high death rate among infants. It seems to me that the matter is largely one of massive infection. As Dr. Stewart pointed out, the infant does have a considerable resistance to the disease.

We have just finished a study of the Mantoux test on all high school children in Rochester, and we found, contrary to some of the figures given for larger cities, an incidence of only 11 per cent. Opie gives as high as 97 per cent of infection in children in Philadelphia.

We studied a special group, in which, through a social service investigator, we had contact with tuberculosis within families. We were surprised to find that in a series of about thirty-four cases only 62 per cent of the children who it was known had come in contact with tuberculous patients responded positively to 0.02 mg. of tuberculin given by intradermal inoculation. We increased the Mantoux dosage to 1 mg. and found that only an additional 6 per cent responded positively to the test.

The other point, just mentioned in passing by Dr. Stewart, is the fact that the lesions of tuberculosis are practically never healed. In so-called healed lesions, Dr. Robertson, our pathologist, constantly points out a small active tubercle at the periphery that shows evidence of recent reaction.

DR. O. M. SCHLOSS, New York: Dr. Stewart's observations have brought out clearly the marked difference between initial tuberculous infections (infections in childhood) and the type of disease found in adults. It seems to be fairly well established that the pulmonary lesions of tuberculosis usually develop on an allergic background provoked by the infection in childhood. In connection with this view, it seems rather significant that the infantile type of tuberculous infection may develop in adults who have not had an initial tuberculous infection and, therefore, no allergy.

DR. B. SCHICK, New York: I was interested in this paper because it confirms all of the statements we made concerning the results of our experience. About twenty years ago I published my studies on the prognosis of tuberculosis in infants, and pointed out that tuberculosis in infants is not as dangerous as Schlossman thought it was. He formerly thought that every infant infected with tuberculosis must die. I remember that among thirty-five cases which I followed up for two years, there were many children who survived. However, it is still true that the earlier the infection occurs and the more massive the infection is, the more serious is the prognosis.

Later, as milder infections could be studied, we learned that many infants, especially those who became infected during the second part of the first year, could recover entirely. The changes in the lung are not so alarming, but there is danger that miliary tuberculosis or tuberculous meningitis may develop in the infant. An infant seldom dies from tuberculosis of the lungs.

As Dr. Schloss mentioned, one has to have infant and childhood infections before the peculiar form of tuberculosis found in adults can develop. During the World War we learned that when soldiers came from the country free from tuberculosis and then became infected, they showed the same picture as that seen in a child: the primary focus and the primary infected lymph glands, similar to what is called the primary complex. Therefore, an adult free from tuberculosis and all symptoms in whom tuberculosis develops, no doubt reacts in the same way as a child.

It is interesting to note that children living in a tuberculous environment frequently escape infection. In studies with Calmette's subcutaneous and intracutaneous vaccination with B C G, we use only children who come from a tuberculous environment. The children must be free from tuberculosis. We have about eighty such children. In our selection we have to be careful, so we test them not only with 1 mg. of tuberculin intracutaneously, but with from 2 to 5 and even 10 mg., in order to be sure of the absence of a tuberculous infection.

DR. J. D. TRASK, New Haven, Conn.: Have you had the opportunity of examining post mortem any of the extensive parenchymatous lesions? It seems obvious that the explanation usually offered for this type of lesion fits in with the current notion of allergy and the effect of dosage. Perhaps this explanation is too easily accepted, and pathologic studies of exactly what occurs would give important results. We found it extremely difficult to recover tubercle bacilli from the sputum of children with extensive parenchymatous lesions similar to those described in the present paper.

DR. F. P. GENGENBACH, Denver: Have your roentgenologists increased the number of diagnoses of tuberculosis or decreased them?

I believe that one or two points brought out by Dr. Stewart are worthy of emphasis: (1) how an extensive lesion may exist in the lung without being demonstrable from the standpoint of physical diagnosis alone, and (2) the large proportion of cases in which the skin gives negative reactions to tuberculin.

I am sure that all of you must find, as we do, that some of the tuberculin on the market is inert. There is no question that a good many persons whose skin seems to give a negative reaction to tuberculin have definite clinical symptoms, and if the provocative method, especially the Mantoux, is used, the reaction becomes positive. In private practice, we now use the Craig test as a routine measure rather than the Pirquet test, as we find it more sensitive. The Craig test is a multiple puncture method similar to that used for vaccination against smallpox. Its results are closer to the percentage of the Mantoux test than are those of the Pirquet test.

DR. B. SCHICK, New York: In connection with the tuberculin solution, the tuberculin I use is always boiled down 50 per cent. If you have a tuberculin solution that is not potent enough to give positive reactions, do not blame the tuberculin reaction, but the impotency of the tuberculin solution. I agree in this respect, entirely with Dr. Gengenbach.

DR. C. A. STEWART: I was interested to know that the incidence of tuberculous infection in Rochester, Minn., was similar to that reported for the rural districts of southern Minnesota by Dr. Slater. Studies on tuberculous infections showed the existence of nests of tuberculosis in certain parts of this country. The logarithmic curve of the incidence of tuberculous infection with increasing age is not a straight line, indicating, therefore, that the additional persons giving positive reactions each year is not directly proportional to the number of persons giving negative reactions in the population at that particular age. The amount of tuberculous infection as it occurs in ordinary human experience is unknown, but

there is evidence that children, in general, successfully resist infections as they ordinarily occur from contact with open cases of the disease, provided reinfection is prevented.

Dr. Helmholz referred to studies indicating that the primary lesion never heals. Some active tubercles persist at the margin of such foci throughout life. A positive reaction to the test with tuberculin, therefore, means that a person has tuberculosis, but does not reveal whether the lesion is of the childhood or of the adult type.

I was interested in what Dr. Schloss said about the initial infection in adults producing lesions similar to those characteristic of the initial infection in childhood. Since the lesions of first infection are similar regardless of the age at which such infections occur, it probably will be preferable to speak of tuberculosis of first infection rather than to use the term childhood tuberculosis to designate this condition.

A study of tuberculous infection in nurses in Minnesota revealed that when they started training, 30 per cent gave a positive reaction to the test with tuberculin, which number increased to 90 per cent before the period of training was finished. This indicates that technic against contagion should be instituted in every general hospital.

In connection with Dr. Schick's statement regarding miliary tuberculosis and meningitis, in such cases we frequently find old calcified lesions of childhood tuberculosis antedating the miliary lesions. I believe it has been found difficult to produce miliary tuberculosis from first infection, but reinfection in a sensitized person produces a different reaction characterized by caseation, toxicity and a lighting up of the disease.

We have not had the opportunity to make postmortem examinations in cases of the childhood type of tuberculosis. Tubercle bacilli have been isolated in one case of this type. This type of tuberculosis has not been considered highly communicable, since for several years we have had several patients with bronchiectasis living among children with the childhood type of tuberculosis, and during this period of contact, tests with tuberculin on the children with bronchiectasis have given negative results.

In answer to Dr. Gengenbach's question, I wish to state that a rereading of the films reduced the incidence of reports of questionable and slight calcification of the intrathoracic lymph nodes. By means of the x-rays, lesions were demonstrated in approximately 25 per cent of persons giving positive reactions to tuberculin. The tuberculin used was obtained from Saranac Lake. Care must be used in applying 0.1 mg. as a routine measure, since pronounced reactions may cause the patient to object to further tests. We have not used a provocative injection, but, in certain instances, in persons giving negative reactions the dosage used in the Mantoux test has been increased to 2 mg. If the reaction remained negative with this dosage, the case was classed as nontuberculous. We have used the puncture method of applying the Pirquet test, and are now making a study of the surface area of the reactions. We know now that the surface area of the reaction is not directly proportional to the amount of tuberculin introduced.

Fourth Session

Evening, April 16

THE SUBSTANCES INVOLVED IN THE COAGULATION OF THE BLOOD OF THE NEWBORN. DR. HEYWORTH N. SANFORD, DR. T. H. GASTEYER and MISS LOIS WYATT (by invitation), Chicago.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

MULTIPLE VISCERAL ANGIOSARCOMAS IN AN INFANT, WITH INVOLVEMENT OF THE LUNG. DR. MARTHA WOLLSTEIN.

This paper was read by title.

THE GROWTH AND RETENTION OF INFANTS RECEIVING EVAPORATED MILK. DR. P. C. JEANS and GENEVIEVE STEARNS (by invitation), Iowa City.

In an endeavor to determine the suitability of evaporated milk for infants, it was fed to a group of babies shortly after birth until they were 10 months old. The milk was purchased in the open market, diluted with equal parts of 12 per cent solution of corn syrup and acidified with lactic acid. This mixture was approximately equivalent in composition to undiluted lactic acid fresh milk with the addition of 6 per cent of corn syrup, which was employed for similar observations on another group of babies during a preceding period. In order to compare the utilization of evaporated milk with that of fresh milk, the quantities of food and the additions to the diet of milk were made the same according to age in the two groups. These quantities and additions have been published in detail (AM. J. DIS. CHILD. **39**:701 [April] 1930).

Clinically, the babies fed evaporated milk did well, with the exception that in the early months the food in the concentration and the amounts administered produced an increased frequency of stools. This symptom was lacking when undiluted fresh milk was fed in the same quantities to the babies of the preceding study. After the first few months, no special difficulties were encountered, and the infants made excellent physical progress at all times. They exceeded the average weight and length of infants in Iowa. They gained as much weight as the group fed fresh milk, and ultimately, though not early, they attained the same body length.

The retention in the early months presumably were affected by the increased frequency of stools. To 4½ months, the retention of nitrogen was somewhat less than with undiluted fresh milk, reaching a low level of 150 mg. per kilogram at 3½ months. However, even this low value is greater than that obtained with a customary formula of a dilution of sweet milk. From 4½ to 8½ months, the retention of nitrogen rose to a high level of from 210 to 240 mg. per kilogram, values considerably greater than those obtained with undiluted fresh milk. After 8½ months, the retention of nitrogen was approximately the same in the two groups.

Before the observations on evaporated milk were begun, the availability of the calcium was in doubt because of publications indicating unfavorable effects on the calcium of milk by prolonged heating. It became evident, however, that the calcium of evaporated milk is readily available to the infant, and that the retention is as great as with undiluted fresh milk, though the curves for retention according to age do not coincide. The average retention for the group varied from 67 mg. per kilogram at 2 months to 46 mg. at 10 months, with a high level of 70 mg. between 7 and 8 months.

The retention of phosphorus roughly paralleled the retention of nitrogen and calcium and was relatively high at all times. From 2½ to 8½ months, the retention was in excess of that obtained with undiluted fresh milk, reaching a high peak of 53 mg. per kilogram at 7 months. The calcium-phosphorus ratio varied from 1.8:1 at 2 months to 1.3:1 at 7 months.

DISCUSSION

DR. O. M. SCHLOSS, New York: It seemed to me that the curves presented by Dr. Jean indicate that the most favorable retention of nitrogen, phosphorus and calcium occurs in the later age, after from the twentieth to the twenty-fifth week. I wonder whether that rise possibly coincided with the beginning of feeding of other substances, such as vegetables or cod liver oil, which were possibly added at that time. Is it altogether attributable to the milk alone? The favorable rise in the peak seemed to occur in the later age, well past the fifth or sixth week.

DR. B. K. HAMILTON, Chicago: Dr. Swanson, who studied the composition of growth by following the progress of breast-fed and of bottle-fed infants for several months, presented a paper before this Society last year. He found that,

chemically, the bottle-fed infants grew in a different way, and that they retained much more mineral in the body than the breast-fed infants, progressing in this way month after month.

The same observations were made previously by Römiger and Meyer of Germany, who studied the retention of ash, while Dr. Swanson analyzed the different minerals. These facts make it clear, in my mind, that if you want to test the value of food, it is not enough to study the retention of various minerals in the formula. If a very good retention is found, it may be that the retention is too good. The retention may be too high when compared with the normal in an infant fed breast milk.

Consequently, I found Dr. Jean's curves for weight and delightful photographs much more convincing than the figures showing a high retention of various minerals in infants fed evaporated milk.

Dr. C. G. Kerley, New York: I have used evaporated milk for feeding infants for many years. I have found it particularly useful in difficult cases. Infants who cannot utilize fresh cow's milk, prepare it as we may, will thrive promptly when given the evaporated product. I have demonstrated this fact in hundreds of infants. There is only one disadvantage, which is peculiar to all heated milk foods and which is overcome by the use of orange juice or tomato juice. In regard to the ultimate results, the infants fed the evaporated product will show, at the sixth or the eighth month, as good physical development or better than those given other food.

Dr. J. Brennemann, Chicago: I have been interested in this subject for some time. In fact, twenty-five years ago, when I had a patient I could not do anything for, I gave him condensed milk.

Evaporated milk, if it can be shown to have the same or approximately the same value as lactic acid milk, has a decided advantage in that one does not have to add anything that might be added in the wrong way, as in cases in which there might be a mistake in the bottle.

The point in which I was particularly interested was this. If I may quote Dr. Jeans, he told me that he did not like evaporated milk. From the presentation he made, I do not see why he does not like evaporated milk. He made one observation I have also made. If one gives the milk in full concentration, half and half in the first week, second or third weeks of life, there is a definite tendency in a certain number of cases to frequency of movements of the bowels. In former years, when I used fresh sweet milk, I always felt that that frequency was a warning signal to make a change. I do not feel that way in regard to evaporated milk. I mean that in a case in which I would ordinarily be worried if there were three or four movements of the bowels, I would not feel the same if evaporated milk were being given. When this milk is used, the irritating, unpleasant, disagreeable and unfavorable features that occur when sweet fresh milk is employed are rarely seen.

Dr. F. P. Gengenbach, Denver: Am I right in supposing that in the series in which diluted milk was used it was not acidified? I think it is rather interesting that as far as the curves for weight and length are concerned the results were about the same with diluted milk as with the concentrated acidified milks, despite the fact that there apparently was less retention of nitrogen, calcium and phosphorus. Was the general condition of the infants equally good in the three series?

Dr. P. C. Jeans: In reply to Dr. Schloss, I do not believe that the increased growth of these babies was due specifically to some accessory food. Accessory foods were added to the diet, as they were also to the diets of the babies with whom this group was compared. Without these additions it is probable that the growth would not have been as good. I believe that the increased growth was due to the large total ingestion of food.

In connection with Dr. Hamilton's remarks, it was of interest to us to compare the retentions of babies fed evaporated milk with those of infants receiving milk

treated in other ways. In view of the question raised in the literature as to whether the calcium of heat-treated milk is as available to the infant as is that of fresh milk, it was also of interest to note that calcium was so well retained.

I wonder if Dr. Brennemann understood that lactic acid was added to the evaporated milk fed to the babies studied. We have under observation a group of babies who are being fed unacidified evaporated milk.

Dr. Gengenbach called attention to the fact that the growth of the infants fed diluted milk was apparently as good as that of those fed undiluted milk, as shown in the charts, though the retention of those fed diluted milk was not as great. One can only speculate in regard to this apparent discrepancy. It might easily be that a larger proportion of the gain in weight in the group given diluted milk was adipose tissue. This conception receives some support in the smaller retentions of nitrogen and calcium in the group given diluted milk.

DR. F. P. GENGENBACH: Was there any difference in the facility with which the infants took either the acidified milk or the evaporated milk? You did not say anything about the quantities taken in either group, but merely showed the curves as to the amount of milk taken.

DR. P. C. JEANS: The quantities of both fresh undiluted lactic acid milk and evaporated milk were the same, namely, about 660 cc. in the early weeks; the amount was increased fairly rapidly until the infants received slightly more than 1,000 cc. at about the age of 6½ months. From that time on, the amount of milk remained constant. The quantities were the same in the two groups, and were made so intentionally.

CHANGES IN THE BONE IN GAUCHER'S DISEASE. DR. KATSUJI KATO (by invitation), Chicago.

Gaucher's disease occurred in three members of a Japanese family living in California, in which the only disease of significance was syphilis in the grandmother of the children on the father's side. The Wassermann reactions of the father and mother were negative. The two older patients showed definite involvement of the bone. The oldest child became crippled at the age of 7 years, with absolute inability to walk toward the end of her life. A roentgenogram made shortly after splenectomy, which was performed three years before death, showed a "worm-eaten" appearance of the shaft of the long bones, owing to the accumulation of Gaucher cells in the marrow, which produced irregular areas of rarefaction and condensation, with a resultant thinning of the cortex. The head of the femur was flattened, its surface was roughened, and the joint surface in general was irregular. The neck of the femur contained many areas of rarefaction, and there was a definite pathologic fracture through the surgical neck on the right side. The entire neck with the head on both sides was bent downward, and the axis was at right angles to the shaft of the bone. The most unique change was seen in the lower third of the femur, where the shaft showed a fusiform expansion, especially noticeable on the medial aspect, so that the normal contour of the bone at this portion, with flaring condyles, was changed to an appearance resembling that of a Ehrlenmyer flask. This interesting change appears to be so constant in all cases of the disease with involvement of the bone that we may regard it as an early pathognomonic sign, especially in the presence of other outstanding clinical features.

The similar changes are demonstrable in the majority of cases in other long bones, especially the humerus, the tibia, the radius, the ulna and the ribs, perhaps in the order named. The pelvis is also affected.

Of the other bones of the body, the most interesting changes are seen in the vertebrae. In 1924, Howland and Rich reported a case before this Society, in which the disease process invaded the lumbar vertebrae rather extensively together with other bones of the body. My cases do not show any vertebral involvement. In all cases reported in which the vertebrae are involved, there is often

a gibus formation with a resultant compression fracture. All the bones of the spinal column are invaded by the pathologic cells, but the intervertebral disks usually remain intact.

The bones of the skull are also affected, though there is only one case on record in which this involvement is demonstrated. Pick gave an illustration of a case, showing the interior of the calvarium, where it was found that the diploe was filled with a mass of Gaucher cells, and the inner surface of the parietal bones contained symmetrically placed yellowish spots of these nests of cells.

A study of cases with involvement of the bone led to the conclusion that in all suspicious cases a roentgenographic examination should never be omitted, for it may be the deciding factor in establishing a correct diagnosis.

Splenectomy is only of temporary value and never produces a cure.

DISCUSSION

DR. J. H. M. KNOX, Baltimore: I think that the Society is fortunate in having this splendid demonstration of the changes in the bones in Gaucher's disease. My interest in this condition dates back about sixteen years, when Dr. Wahl, Dr. Schmeisser and I reported two cases in infants which we considered Gaucher's disease. One infant came under observation at the age of 9 months and died at 11 months; the other was first seen at the age of 5 months and lived to be 15 months of age. The infants were sisters, being born one after the other of Russian Jewish parents; they lived in Baltimore.

The case of the youngest patient described up to that time was reported by Niemann in 1914. The infant had been ailing since the age of 2 months, and had died at 17 months with symptoms and microscopic changes apparently identical with those in the cases we described. Most of the cases reported in the literature have occurred in later childhood or adolescence. About two or three of the patients have lived to be 30 or 40 years of age.

In our cases there was an almost general distribution of the large, pale, granular or finely vacuolated cell containing material of a lipoid character, but not giving a chemical reaction to fat and perhaps resembling lecithin. I do not believe that the exact chemical character of this material has as yet been determined. It would seem that the severity of the condition is more marked in infancy; more tissues are involved, and there is a larger number throughout the body of the characteristic cells. We found typical cells in the bone marrow on smears, but we did not procure sections of the bone, and we did not notice a deformity, as our patients had never walked. Our second case was diagnosed by finding the characteristic cells in the lymph gland excised before death.

Another interesting observation was made in the second case. On ophthalmologic examination, cherry-red spots similar to those described in amaurotic family idiocy were found. In certain of these cases cells have been described which resemble those found in Gaucher's disease. I have wondered whether future study might not establish some connection between the two conditions. This whole group of diseases is of extreme interest, perhaps especially to the biochemist. The characteristic cells apparently originate in the reticular and endothelial tissues. The sallow complexion and the enormous enlargement of the liver and spleen, with symptoms of gradual loss of weight and exhaustion and a blood picture of secondary anemia, should lead one to suspect and to investigate the presence of cells characteristic of Gaucher's disease.

DR. F. W. SCHLUTZ, Chicago: I believe that the experience of Dr. Kato is unusual; to find this extremely rare condition in three members of the same family is unique. I have never observed such a case.

DR. K. KATO: The point that Dr. Knox raised as to the possible relation of amaurotic family idiocy and Gaucher's disease, and also the condition generally known as xanthomatosis, as brought out not long ago by Rowland, is interesting. As soon as we know more about the functions and anomalies of the reticuloendothelial system, we may be able to correlate these diseases, all of which are characterized by the deposition of various substances in the macrophages.

TUBERCULOSIS IN CHILDHOOD FROM A CLINICAL STANDPOINT. DR. A. ABALLI.

This paper was read by title.

A SYMPTOM COMPLEX: ANOREXIA, GASTRIC RETENTION, MUCOUS GASTRITIS, INTESTINAL STASIS. DR. CHARLES G. KERLEY, New York.

Every patient presented because of anorexia is given a complete physical examination in order to exclude ailments other than gastro-intestinal disturbances. In this examination we have found it essential to pay particular attention to the upper respiratory tract. Chronically infected tonsils and adenoids and sinusitis may be potent factors in the loss of appetite. In the majority of cases, simple procedures are effective in the treatment for failure of appetite. A suitable selection of food given at intervals of from four and one-half to five hours without entertainment or urging, together with the establishment of normal elimination, fulfils the requirements.

During the past four years, these measures have failed in 535 patients, ranging in age from a few months to 10 years: In 270 cases, the patient was the only child; in 172, there were 2 children; in 61, 3 children; in 24, 4 children; in 1, 5 children; in 3, 6 children, and in 4, an undetermined number.

In this group our procedure was to give a normal breakfast; in the group of bottle-fed infants, a milk formula. For infants who were able to run about and older children, the meal consisted of cereal or an egg, milk, bread and butter. Five hours after the completion of the meal, no water having been given, a tube was passed into the stomach to determine the presence of residue of food, mucus or both. We have found the outstanding cause of anorexia in the resistant cases to be due to the presence of a gastric content when the stomach should be empty. We have demonstrated this failure in function to be due to inertia of the stomach. This condition has been found in the ptosed stomach, usually associated with mucous gastritis. The most frequent cause of delayed emptying occurs in association with intestinal stasis apparently dependent on a habitually spastic colon or on an elongated so-called redundant colon and sigmoid, an anomaly that indirectly encourages spasticity. We also observed the tendency to spasticity and the absence of an anomaly in the highly neurotic, so-called spasmophilic child with constitutional nerve imbalance, which in some cases appears to be emphasized in the intestinal tract. We further found that this abnormality in function may be occasioned by irritant agencies, such as retained feces, in the intestine, as in chronic appendicitis.

We found gastric retention due to pyloric spasm in a few cases. In these cases there is apt to be abdominal pain, vomiting and eructations of gas. Presumably, acute or subacute gastritis is present.

An abdominal belt was employed in cases in which ptosis occurred. Lavage was carried out in those in which mucous gastritis was found. Aromatic fluid extract of cascara sagrada was given three times a day in a dosage sufficient to produce two evacuations daily. The sphincter ani was investigated and stretched, if necessary. Atropine in near physiologic dosage was given in conjunction with a simple alkaline powder.

For bottle-fed infants, the interval between feedings was never less than four hours; for, older children, it was five hours. Skimmed milk was always used, and cream and butter were omitted entirely. In my most troublesome cases, the anorexia was due primarily to intestinal stasis, delayed emptying of the bowel and habitual constipation.

A consideration of gastro-intestinal disorders in which anorexia is a prominent symptom shows that we are inclined to look on the various divisions of the gastro-intestinal system as independent. The various divisions have, in a measure, a special function, but for proper functioning of the entire gastro-intestinal tract from the esophagus to the sphincter ani there is decided interdependence. These lantern slides show the belt worn by patients with ptosis, gastroptosis, mucous gastritis, gastric retention, pyloric spasm, a spastic colon, colonic stasis and an elongated prolapsed colon and sigmoid.

Fifth Session

Friday Morning, April 17

THE INFANT WARD. DR. JOSEPH BRENNEMANN, Chicago.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

DISCUSSION

DR. B. SCHICK, New York: Dr. Brenneman expressed, so to speak, the same ideas that I express to my staff. I have all the infants placed in cubicles and do not permit an unnecessary number of the staff to enter the cubicles; they have to stand outside. I point out, as Dr. Brennemann did, that it is not so important to know whether there are two or three more pneumonic foci on the right or the left side of the lung; it is more important that the children should not be overexamined.

Flexner studied experimental epidemics in mice villages. The observations can be seen in infants' wards. I do not like to move a child from one cubicle to another or from one ward to another, because the child may become infected with new germs in the place to which he has been moved.

I have frequently fought against the conditions in the nurseries of maternity wards. In such a nursery, you see the newly born children, like loaves of bread, one near the other. A physician told me that he visited such a nursery and saw a child with vomitus on his diaper or his clothes. He asked the nurse whether this child had vomited. The nurse said no, but that the child above had vomited.

I do not know how to change the situation in the nursery. I visited a nursery in New York where there were thirty-four new-born infants in a room that was no larger than a corner of this room; there were not even enough cribs, but drawers from the desk were used to put the infants in. As I left the place, another infant was brought in. If an estimate of the consumption of oxygen and the production of carbon dioxide of all these infants, including from four to six nurses and the physicians, were made, one would find the overcrowding worse than in the slums.

DR. J. L. MORSE, Boston: For the last few months I have been occupied with looking up the history of pediatrics in Massachusetts for the anniversary meeting next June. I was much impressed that there is nothing new under the sun. The mortality in foundling asylums in Massachusetts was, I think, about 85 per cent. Dr. Putnam, who was a member of this Society years ago, established the so-called Massachusetts Infant Asylum in 1867. From 500 to 1,000 babies were taken into this asylum each year. There were something like twenty beds. Never more than half of the beds were supposed to be occupied. Wet nurses were employed, and every baby under 3 months of age was partly breast-fed. The babies were sent to foster-homes in the country as soon as possible.

Through the eighties the mortality was about 10 per cent, and during the early nineties, it was only 5 per cent.

It seems to me that Dr. Putnam discovered everything about which we have been talking for the last ten or fifteen years.

DR. I. A. ABT, Chicago: In hospitals, infant morbidity and mortality depends on the management in the wards and the care of the individual baby. The reduction of infant morbidity and mortality is due to the advances in hygiene in hospitals and in communities.

Dr. Schick told about the 104 per cent mortality in Prague; the same percentage occurred in Berlin until Finkelstein took charge of the Kinderasyl. The improvement in the conditions in New York may be ascribed to similar causes.

A system of aseptic nursing and care has been evolved, but it is not in general use.

DR. H. C. CARPENTER, Philadelphia: Infants are frequently kept in the ward too long. They should be transferred to the outpatient clinic as quickly as possible. For most infants, a well organized outpatient clinic is better than either the ward or the foster-home. In such a clinic adequate time is allocated for thorough medical study. It provides efficient social service work under medical direction and visitation in the home by field workers who are registered nurses with social training. The patient's home is visited the day following its admission to the ward, and a social diagnosis is made and attached to the history kept in the ward. Every effort should be made to prepare the home for the early return of the child. After admission to the outpatient clinic, repeated visits are made to the home by a field worker, who, acting as a health teacher, encourages the mother to carry out effectively the directions given by the physician. The health education of the mother by the nurse in the home is the best means of preventing the infant's readmission to the ward.

DR. C. G. GRULEE, Chicago: I do not like to sound a discordant note in this beautiful symphony, but it seems to me that there are certain things to be said on the other side.

In the first place, owing to an advance in pediatrics, we probably save more premature infants than previously. These infants are placed in the wards, and they are notably peculiarly susceptible to infection of the respiratory tract resulting in death.

Another experience that I have had repeatedly was not taken into consideration in Dr. Brennemann's paper. An infant leaves the hospital in excellent condition from the standpoint of nutrition and without an infectious disease. Within a week or two, that child comes back to the hospital in a dying condition. In these cases, the infection of the respiratory tract is acquired in the home.

It seems to me that a great deal depends on the head nurse. I think that the whole atmosphere of the wards is determined by the head nurse and her attitude toward the babies.

A second point that I wish to bring out is that in many hospitals there are not sufficient nurses to give proper care to the infants. I do not think that, without considering the subject carefully, we should blame the conditions on the infant wards for a number of matters that probably would be just as bad, if not worse, if the children were placed in the home.

Another point is that investigators who compare the infant ward with the home compare infants from very poor homes and very poor surroundings with those who live in homes with good surroundings and good hygienic conditions.

There is a great deal to be said on both sides, and I should hate to have the whole subject pass without uttering a protest against such a damaging statement as has been given.

DR. J. B. BILDERBACK, Portland, Ore.: I do not hold a brief for the institutional care of children of any age. Undoubtedly, homes in which there is proper cooperation and environment are the best. For two years there were no deaths in the Waverly Baby Home in Portland, Ore., where there were seventy-five or eighty babies, most of them under a year old. Last year two infants died, one from a malformation of the genito-urinary tract; the other was a premature syphilitic child. We are probably favored by the conditions in the spacious baby home, where the infants are kept outdoors in the sunshine a great deal, with close supervision of feeding.

I refer particularly to baby homes. Our problem in the infant ward in the children's hospital is the same as Dr. Brennemann pointed out. We think that a baby should spend the shortest possible time in the ward. Babies who have harelip or cleft palate are our most difficult problem, as they are prone to infection. An ideal method would be to have the infants brought in for operation and the work done immediately; they should then be sent away from the hospital for convalescent care.

DR. J. BRENNEMANN: Dr. Schick spoke of cubicles. In my opinion, they have been rather disappointing. It might be said, as Dr. Abt suggested, that you do not take care of your infants the way you should. Perhaps your nurses are not doing everything they should and perhaps you are not using cubicles the way you should. A few years ago I visited a hospital in which cubiculization and all procedures that pertained to cross-infection, etc., were carried out to the most extreme, meticulous extent. There were different cards on the beds indicating the different degrees of care one should exercise in handling each patient.

I exercised the privilege, which I think one always has in going to a hospital, of not asking the head physician how things were, but I went to the one man who knows the situation best, the resident physician. I said, "I notice you have cubicles. What do you think of them?"

He said, "I have been here a year and I do not know yet. I think they are very protective in diphtheria and scarlet fever. I do not think they do much good in measles and chicken pox." I asked him about infections of the respiratory tract, and he said that he did not think the cubicles did any good in such cases. That pretty nearly expresses the situation.

In my original paper I suggested that one ought to have at least one nurse to every two babies. We have figured out that we have a nurse to every one and seventy-five-hundreths of a baby.

I would like to make one last remark in connection with a point that has always impressed me very much, a remark that Dr. Chapin made to me some years ago. As you know, he has done wonderful work in connection with the Speedwell plan of foster-homes. He said, "I am devoting the rest of my life to making up for the wrong I did in sending babies to infants wards and taking care of them there." That statement may be a little strong, but it contains good sense and has a great deal of heart back of it. I think that Dr. Abt emphasized an important point, that one should have one's heart in the matter also.

THE EFFECT OF THYROID MEDICATION ON THE ENERGY METABOLISM OF NORMAL INFANTS. DR. T. C. WYATT (by invitation), Winterville, N. C., DR. L. E. WEYMULLER (by invitation), Brooklyn and DR. S. Z. LEVINE, Flushing, N. Y.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

A REVALUATION OF THE RÔLE OF SINUSITIS IN THE DISEASES OF CHILDHOOD. DR. EDGAR J. HUENEKENS, Minneapolis.

The most rational indications for tonsillectomy are a history of repeated infections of the upper respiratory tract or the finding of a general condition due to a focus of infection. The local appearance of the tonsil and its size are of comparatively slight importance.

In Kaiser's latest report on the results of 4,000 tonsillectomies, he came to a qualified approval of the ultimate results, but his conclusions should not be considered the final word on the removal of focal infection, because he completely ignored the sinuses and because the statistical method does not properly emphasize outstanding success in individual cases.

The indication for surgical intervention in the nasal sinuses is the same as that for tonsillectomy.

Roentgen examination is the best available method for determining the condition of the sinuses, but it cannot be relied on altogether. The history of the case and the results of a thorough physical examination must finally determine this question.

In this study, the maxillary sinuses alone are considered, because they are easily accessible and are often the first part infected; also, irrigation of them is harmless.

In our series, repeated "colds" and infections were noted in 93 of 122 successive cases. In 49 cases, there were general conditions aside from the local symptoms, which indicated the reason for surgical intervention:

Endocarditis	7
Rheumatic fever	6
Nephrosis	5
Nephritis	4
Asthma	4
Malnutrition	4
Secondary anemia	4
Pyelitis	3
Bronchiectasis	3
Persistent râles	2
Chorea	2
Recurrent abdominal pain	2
Indefinite rheumatic pain	2
Purpura hemorrhagica	1
	49

A comparison of the roentgen and operative observations is as follows:

Pus found in the sinuses of		60
Pus not found in the sinuses of		62
		122
X-ray (—)	Operative observations (—)	20
X-ray (—)	Operative observations (+)	8
X-ray (+)	Operative observations (—)	39
X-ray unsatisfactory		2
X-ray not taken		2
X-ray (+)	Operative observations (+)	51
		122

In the 51 cases in which roentgenograms showed infection of the sinuses, the following discrepancies were found:

Roentgen observation of bilateral sinusitis with unilateral operative observations	12
Unilateral roentgen observations, with bilateral operation changes	7
Roentgen observations of sinusitis on left, with operative changes on right	1
	20

Irrigation of the antrums is not intended to replace tonsillectomy, but it is an added means of removing common focal infections. In children, irrigation of the antrum with the establishment of a window is a comparatively harmless procedure, and in many cases, owing to the favorable anatomic situation of the antrum, it need be performed only once.

DISCUSSION

DR. C. G. KERLEY, New York: Dr. Huenekens has given a most timely contribution, and I am sorry that he had to hurry to complete the demonstration. I now make an examination of the sinuses as a routine procedure in cases of frequent colds, asthma, obscure muscular pains and when there is a persistent slight elevation of the temperature. I found the referring of cases for examination so unsatisfactory that I took over the work in my own laboratory. We had considerable trouble at first in establishing a standard of normal, the period of development and the appearance of the sinuses in disease. There are little or no data concerning the sinus and its development in children. At the present time we are making a roentgen study of as many of our infants under 1 year of age as we can handle with the view of learning something regarding the development of the sinuses and when they may become the site of an infection.

To the present time, we have found involvement of the maxillary sinus in a number of cases, in agreement with Dr. Hueneken's observation; the ethmoids have been slightly involved in comparatively few patients.

DR. J. BRENNEMANN, Chicago: Dr. Huenekens has goaded me into discussing his paper. I have had a hard time deciding whether I have been a poor observer all my life or whether I am beginning to suffer from senile conservatism. In my

own experience, focal infections have played an insignificant part in connection with distant pathologic changes in children.

What do we mean by focal infection? If we mean acute tonsillitis or acute infection in some other part of the body, I am willing to admit that it does a great deal of harm. If, on the other hand, we mean a persistent or slumbering process in the sinus or the mastoid, or some other condition of that sort, especially in the teeth, in my experience I have rarely been able to see the serious results or complications ordinarily attributed to them.

There are several interesting points in reference to the antrum. In the first place, it has always been my idea, and it has been carried out in my experience, that with every severe infection of the nose and throat the antrum is apt to be involved, just as the mastoid antrum is involved in otitis media. I am positive that I have had an infection of the antrum twenty-five times in my life, and I have never consulted an otolaryngologist about it. If at such times I had had roentgenograms taken, I believe they would have shown a definite clouding of one or more of the sinuses.

I saw a child with a clouded sinus on one side who was the picture of health, but who was said to have what is known as a "sinus lung," if you know what that is. I do not. I referred her to a laryngologist. Before he had finished, he had also fitted her with glasses and rather insisted on an operation. I exercised my conservatism, and indicated to the mother that since the patient was in good health we had better take another roentgenogram at a later time. When the roentgenogram was taken, it showed a normal picture. I do not believe that the clouding of the sinus had anything to do with her condition, but was an accompaniment of occasional infections.

I wish to mention one other point in reference to antrums and sinuses. Once in a while I read a paper by one of the investigators who have brought the sinus and antrum in particular into special prominence. I recently read a paper by Dr. Dean, and I thought, "I will now find out exactly what they do to cure diseases of the antrums." When I came to the part that dealt with therapy, the outstanding statement was that one must be very conservative and not do much. In this point of view we were on a basis of perfect agreement. My experience has been that the sinuses of children rarely require treatment. Possibly we are influenced by our own experiences. I notice that Dr. Huenekens has had five patients with nephrosis treated in that way. For a short period, when a child with nephrosis did not get along well, I allowed myself to think that possibly the condition might be due to an infection of the antrum. In one case I permitted a specialist in diseases of the nose and throat to look at the patient. He put a window in the antrum. Pneumococcus septicemia developed, and the child died a short time afterward.

For fifteen or twenty years, I have expressed the opinion that the great majority of cases of pyuria occur in connection with acute respiratory infections. I have never seen a patient with pyuria of long duration get well because a focus of infection was removed. If you remove a focus of infection, the patient will get well, but not, I believe, because the focus of infection was removed. I would even extend the remark to cases of acute hemorrhagic nephritis.

DR. A. F. HARTMANN, St. Louis: I wish to say a word for the specialist in diseases of the nose and throat. Before Dr. Dean came to St. Louis, the pediatrician was usually responsible for the diagnosis of infection of the sinus or of the mastoid, and it was the pediatrician who asked the specialist to treat these infections. The pediatrician really thought he knew more about the nose and throat than the specialist. After Dr. Dean came to St. Louis, we learned a great deal more than we knew before.

In the first place, in an effort to clean the nose and throat, we used to be satisfied with removal of the pharyngeal tonsils, or adenoids and the faucial tonsils, with irrigation of the maxillary antrums and, if thought necessary, the making of windows in such antrums. We were encouraged with the results obtained with these efforts. However, we also were frequently disappointed in

that in many cases the patients did not seem to improve as much as they should, particularly the group with hemorrhagic nephritis, the symptoms of nephritis lasting longer than four or five months.

Dr. Dean pointed out clearly, however, that there could be, and often is, much pathologic lymphoid tissue, in addition to the infected pharyngeal and faucial tonsils, and he demonstrated that the lingual tonsil is sometimes enormously hypertrophied and severely infected, and that the lymphoid tissue on the posterior wall is also a possible source of infection. Since we have been removing more thoroughly all of the infected tissue possible, we think that we have been getting better results.

Dr. Dean also pointed out that the infection of the middle ear may be due not so much to direct extension through the eustachian tube as to extension in the mucous membrane and in the submucosa.

DR. A. G. MITCHELL, Cincinnati: Dr. Huenekens said that this was a controversial subject and I believe it is. I noticed that in his work the subject of tonsillectomy was involved. I wish to mention that phase of the subject.

On three separate occasions and over a period of years, I have tried seriously to study the results of tonsillectomy and to get my mind clear on the indications for this operation. I came to the conclusion that I could not solve these problems to my satisfaction, and this naturally led me to examine the literature a little more carefully than I had done previously. I have come to believe that the indications for tonsillectomy and the results that come from it must be solved in some way other than the statistical method. There are many factors entering into the subject that are not taken into account as they should be. For example, the age at which tonsillectomy is performed is a matter that could be studied, but is one that has hardly ever been mentioned in the evaluation of results. In certain diseases, such as the group of rheumatic manifestations, the curve of incidence is hardly influenced by tonsillectomy. Some factors, such as the effect of season, exposure to infections, and the like, are most difficult to analyze.

The most important point that I believe cannot be evaluated is the accuracy with which histories are secured. In other words, we might carefully analyze data that were received from sources that render it unreliable.

DR. E. J. HUENEKENS: I am interested in what Dr. Brennemann said about roentgen examination of his own sinuses. He probably has had sinus trouble many times in his life. I believe that every one has. Perhaps I have not made it clear that I am talking only about chronic conditions of the sinuses. He mentioned the case of a child who had a cough coincident with sinusitis, and he said that washing out the sinus would not stop the cough. I do not think that he is qualified to make such a statement until he has tried the procedure; he is only speculating. If he had tried it, I would accept his word more readily than I do now.

The case I reported was a similar one, in which the child had been coughing for two and one-half months. Within twenty-four hours after we had washed out the sinus, the cough had stopped. That does not necessarily prove anything, but when you have a number of such cases it is a queer coincidence.

Dr. Hartmann mentioned one specialist in diseases of the nose and throat in particular, and I mentioned the same one in my paper. I am at the stage they were in St. Louis a number of years ago. I have to decide these matters for myself, and I feel hesitant in doing so. If we do not do it, we have no one to do it for us, and we are in a position where we feel that something should be done. If there were more men like Dr. Dean, I would be glad to resign my responsibility.

Dr. Mitchell said something about histories of previous infections of the nose and throat. I think I mentioned in my paper that one of the important points was, instead of obtaining a history, by personal observation of the physician to determine the number of infections and their severity. For that one reason, I believe that a pediatrician is in a better position to determine the indications than a specialist in diseases of the nose and throat, because he has had the opportunity of personally watching the patients.

Dr. Brennemann said something about nephrosis. We have had good results in removing focal infections in a few cases, but, in general, they have not been satisfactory. We were experimenting with this procedure to see the results. In one case in particular, that of a child with nephrosis of one and one-half years' duration, the condition cleared up after removal of the focal infection; however, in the majority of cases, our results were not good.

DR. J. BRENNEMANN: May I have the privilege of correcting the impression I apparently left? I did not for a moment mean to say anything about Dr. Dean that required a defense from Dr. Hartmann. I have profound respect for him. The only reason I mentioned him was because I read a paper in which he and I were in thorough agreement as far as treatment was concerned.

THE MECHANISM OF ANEMIA IN INFANCY. DR. HUGH WILSON JOSEPHS, Baltimore.

The state of the blood at any time is to be considered as the result of an equilibrium between the rate of the formation and that of the destruction of blood. This work was an attempt to study the equilibrium quantitatively. An estimate of the rate of the formation of blood was obtained from the reticulocyte count, of the destruction of blood, from the total excretion of urobilin in the stools, leaving out of account that of the urine, which constitutes only a small proportion of the total. It was found that in normal cases the excretion of urobilin was roughly proportional to the total amount of hemoglobin present.

$$\frac{\text{1 c. urobilin excretion}}{\text{blood vol.} \times \text{hgb. per 100 cc. blood}} = \text{a figure that might properly be called the index}$$

of the destruction of blood. The volume of blood was taken roughly as 0.076 times the weight. On using this method of calculation, the index of the destruction of blood was normally found to be about 5 or 6 mg. of the excretion of urobilin for each 10 Gm. of hemoglobin in the body. In normal cases, the reticulocyte count was about 0.8 per cent. Thus, it may be seen that a rate of the formation of blood indicated by a reticulocyte count of 0.8 per cent balances the rate of the destruction of blood indicated by an index of 5 or 6.

This equilibrium was indicated on the chart by columns of equal height. It was shown that as one column overbalanced the other during the course of pneumonia and convalescence, the hemoglobin fell or rose according to whether the column for the destruction of blood overbalanced the column for the formation of blood, or vice versa. On using this method, it was found that the formation of blood had to be considered in two phases: (1) hematopoietic activity or stimulation and (2) the synthesis of hemoglobin. This was shown in two cases by the effect of iron in immediately causing a rise in red cells coincident with a rise in reticulocytes, whereas the hemoglobin was little affected until after the administration of copper, which appeared to have a specific effect on the synthesis of hemoglobin.

DISCUSSION

DR. I. A. ABT, Chicago: Could you tell us what becomes of the red cells when the hemoglobin falls?

DR. H. W. JOSEPHS: I have followed only the change in the hemoglobin, but from the report the red cells fall at about the same rate.

DR. J. BRENNEMANN, Chicago: I wish to ask Dr. Josephs about congenital aplastic anemia. My impression was that in this condition the blood was destroyed rather rapidly, and my understanding was that if the infant was given a transfusion at the age of 4 or 6 weeks, profound anemia would have developed long before that period.

DR. H. W. JOSEPHS: The highest reading for hemoglobin the patient ever had was around 35 per cent after transfusion. A second transfusion would have made a higher reading, but the destruction of blood would have been greater. Had I charted the readings for the destruction of blood as I did in the other cases, they

would have been about normal, except after transfusion, when the rate was a little above normal. Ordinarily, the rate of the destruction of blood was 5 or 6 units per ten grams of hemoglobin, as it is normally.

THE POSSIBLE SIGNIFICANCE OF CHANGES IN BLOOD LIPIDS IN EPILEPSY. DR. IRVINE MCQUARRIE, Minneapolis, W. R. BLOOR (by invitation), DR. H. C. PATTERSON (by invitation) and MISS CLARA HUSTED (by invitation), Rochester, N. Y.

Because of the recognized importance of lipids, such as lecithin and cholesterol, in the physiology of all cells and because of the characteristically large amounts of these compounds in the tissue of the brain, it appears to us that any definite data indicating reversal of the normal relationships between them may be accepted as presumptive evidence of disturbed physiology of the cells. On the assumption that alterations in the tissue lipids might be indicated by changes in the blood, a rather extensive study of the lecithin, cholesterol and total fatty acids was conducted in 1926 and 1927, as part of a comprehensive investigation into the metabolism of epilepsy in children.

The results may be summarized as follows: In the 130 cases studied, no definite evidence was found to support the opinion that there is a threshold level of cholesterol in the blood below which convulsions occur. Certain patients had seizures in spite of values for plasma cholesterol that had been elevated by dietary measures to from 50 to 100 per cent above the normal level. What is probably a significant finding, however, was a close correlation between the magnitude of the lecithin to the cholesterol ratio in the blood and the occurrence of seizures. When from five to seven samples of blood were taken serially throughout the day from twelve severely epileptic patients, the ratios, in almost every instance, were found to be from 25 to 100 per cent higher during the period of the day when seizures occurred than at other times. In one patient, who was kept on a ketogenic diet for several months with improvement but without complete cessation of seizures, determinations were made at intervals under uniform conditions. In the series of nineteen complete determinations of lipid, there was a definite gradation in the lecithin-cholesterol ratio, the highest being found nearest to the time of a seizure and the lowest most remote from the time of seizures. When the effects of various dietary regimens were studied under well controlled conditions in a severely epileptic girl over a period of sixty-three days, it was found that the lecithin-cholesterol ratio varied from 0.84 during a six-day period of freedom from seizures to 2.09 five days later, when seizures began to occur with great frequency following a radical change in diet. Sudden changes from a ketogenic to a nonketogenic diet or from a ketogenic diet with an acid ash to a similar diet with an alkaline ash caused an increase in the ratio and the recurrence of seizures. In some instances the increases in the ratio appeared to have preceded the convulsions with a tendency to reversal afterward, but in most instances it was impossible to determine with certainty the time relationship between the two.

Since it is generally accepted that lecithin is hygroscopic and tends to increase the permeability of the structures of cells to water and electrolytes, whereas cholesterol has the opposite effect, we believe that the observed imbalance indicates an abnormal permeability of the cells associated with the convulsions of epilepsy. This conviction is strengthened by our finding, as reported elsewhere, that the exchange of water and mineral in the cells may often be demonstrated to be disturbed under similar conditions. The tentative conclusion that genuine epilepsy is characterized by an instability in the mechanisms governing permeability and adsorption of the cellular membrane is supported by the fact that all factors that increase permeability, such as prolonged pituitary antidiuresis, anoxemia and alkalosis, tend to cause convulsions far more readily in these patients than in normal subjects. The possible bearing of these results on the convulsive phenomena in other conditions is discussed. It is fairly generally conceded that there is a high degree of correlation between the metabolic activity and the irritability of any type of tissue on the one hand and its content of both water and phospholipid on

the other. While it is already known in a general way that the water content of the brain is highest and the cholesterol lowest during early life, when convulsions are most frequent, there is great need for further direct analytic study of the tissue of the brain of epileptic patients and of other patients subject to convulsions.

DISCUSSION

DR. S. W. CLAUSEN, Rochester, N. Y.: Have you been able to observe an increased ratio preceding convulsions? Since the lipid in the blood is readily influenced by such factors as diet and starvation, it seems possible that convulsions might have some influence in causing an outpouring of lecithin at a greater rate than cholesterol.

DR. I. MCQUARRIE: In going over the data, we found that that happens in some cases, just as the potassium diminishes before a convulsion. There seems to be some change when the patient has an aura. From the chart it can be seen that some of the determinations showed a falling off in ratio after the convulsion. The difficulty with the study is that you can get samples after the patient has had convulsions, but you cannot anticipate a convulsion. We have not ruled out the effect of convulsions, but we have determinations which indicate that one has already occurred. When the patients are in a very reactive state, during which they have frequent convulsions, you find the same change.

POSTINFECTIOUS ENCEPHALITIS AND MYELITIS IN CHILDREN. DR. T. C. HEMPELMANN, St. Louis.

Postinfectious encephalitis and encephalomyelitis has appeared to be on the increase in recent years, especially following vaccination against measles and smallpox. The disease is apparently quite distinct from the epidemic ("lethargic") type, but there is reason to believe that the pathologic picture is similar in the acute encephalitis of measles, after vaccination and in other acute infectious diseases.

Of thirty-eight cases of postinfectious encephalitis and encephalomyelitis, fifteen were seen in the past year and twenty-six in the last three years. Of these, measles constituted eleven cases; mild infections of the upper respiratory tract (influenza?), twelve; pertussis, four; scarlet fever, two; mumps, two; lobar pneumonia, one; pyelitis, one, and vaccination against smallpox, five. Among the fifteen cases seen in the past year, measles caused nine and vaccination three.

The onset was invariably sudden, with headache, vomiting and, occasionally, convulsions, and the most striking symptoms were profound lethargy, accompanied in many instances by paralytic symptoms (flaccid or spastic), tremor, aphasia and mild or transient meningeal symptoms. The latter consisted of slight rigidity of the neck and occasional Kernig, Babinski or other pathologic reflexes that persisted for only a few days. In most instances, the spinal fluid showed a slight increase in globulin and cellular elements. In a few instances, the cells in the spinal fluid numbered as high as 400 or 600, with a great preponderance of mononuclears.

In the cases of measles the encephalitic symptoms appeared between the second and fifth day after the initial appearance of the rash, and in the cases following an infection of the upper respiratory tract, between the first and tenth day after the onset of the respiratory symptoms. Three of the postvaccinal cases occurred from ten to fourteen days, one four days, and one three weeks, after vaccination. The ages of the patients with measles ranged from 4 to 10 years; of those with infections of the upper respiratory tract, from 1 to 14 years, and of those with postvaccinal symptoms, from 5 to 7 years.

Only three of the thirty-eight patients died. Twenty-nine recovered completely, a few within two or three days, most of them within from ten to fourteen days and a few within two or two and one-half months. Of the six whose ultimate fate is still uncertain, one left the hospital on the second day and could not be traced, one was practically well in three weeks, but still had some visual defect, two showed incomplete recovery in one month, and two showed incomplete recovery in two and three-fourths months.

DISCUSSION

DR. F. C. NEFF, Kansas City, Mo.: I wish to mention briefly two cases of encephalitis that I observed this winter at our hospital. One occurred in a child who, during an active eruption of chickenpox, was brought to the hospital with a condition that was supposed to be tuberculous meningitis. As we knew the occasional occurrence of encephalitis due to chickenpox, we were not surprised when this proved to be the diagnosis. The two interesting features in this case were that it was of the meningeal type, with stupor and definite rigidity of the neck, and that the changes in the spinal fluid were such as one would expect to find early in cases of tuberculous meningitis. The child was well within the course of two or three weeks.

The other case occurred in a child following an unsuccessful vaccination against smallpox. It appeared two weeks after the vaccination. I had no way of finding out what lot of vaccine had been used. Following a typical course of treatment for encephalitis, the child made a definite improvement and finally recovered.

DR. J. BRENNEMANN, Chicago: It seems to me that in the past we have been in a muddle concerning encephalitis, but that the problem is being cleared up by such reports as that of Dr. Hempelmann. We surely did not see as many cases of encephalitis in my earlier years as we do now. We certainly did not see cases such as the one Dr. Hempelmann has just reported.

About twenty years ago, Dr. Abt wrote a paper on encephalitis, and the rest of us wondered what he was talking about. For a long time I had the general idea that if I saw a child with neurologic symptoms of a peculiar nature it had tuberculous meningitis; if the child got well, the case was one of encephalitis.

The word epidemic encephalitis has irritated me somewhat, not today, but ordinarily. At different times, we have seen manifestations of this sort. In 1918, there were many cases of sleeping sickness, a peculiar epidemic. Once in a while we still see cases that grossly resemble that sort of case, and they possibly do belong in the same category.

In regard to epidemic encephalitis, I doubt whether Dr. Hempelmann, if he was as active some years ago as he is now, saw as many cases at that time as he has seen recently during epidemics of measles.

Until three years ago, I had never seen encephalitis in measles, mumps, whooping cough or chickenpox. There are so many cases of encephalitis following other diseases that it is with us constantly.

I wish to ask Dr. Hempelmann about the sugar content of the spinal fluid in his cases. We have come to look on the high sugar content of the spinal fluid as an almost unfailing symptom of encephalitis in a questionable case, and it has great diagnostic value in contrast to the low count one obtains in all types of meningitis.

To my mind, encephalitis is a complication of infection. The most common infection, in comparison with which all others are insignificant, is an infection of the upper respiratory tract. Therefore, I think that there are more cases of encephalitis accompanying infections of the upper respiratory tract, frequently mild and sometimes not, than accompanying any other type of infection. However, I think that involvement of the brain may accompany any type of infection, whether it is a definite infection, a bacterial invasion or, perhaps, merely a toxic condition.

DR. I. A. ABT, Chicago: Dr. Hempelmann's paper suggests some historical references. In 1865, Virchow described encephalitis of the new-born. His finding was confirmed by some investigators and rejected by others. It may have been postmortem softening. In 1876, Huguenin showed that encephalitis was an independent disease, and he differentiated it from abscess of the brain. In 1881, Wernicke described a form of encephalitis that occurred among alcoholic patients, and somewhat later Strümpell described poliencephalitis anterior. Strümpell thought that this sort of inflammation of the brain was related in some way to poliomyelitis. Later on, the designation suggested by Strümpell was dropped, and the term "encephalitis of children" was accepted. Freud and Rie described

numerous cases of their own. They pointed out that poliomyelitis and encephalitis were not related diseases, and they showed that encephalitis occurred after a variety of acute infectious diseases.

Measles is at the top of the list of causes. Scarlet fever, variola and pertussis have been described as causes by Henoch and Osler, Gowers and Finlayson. Pertussis has recently received attention as a frequent cause of encephalitis in some publications of the *Monatsschrift für Kinderheilkunde,* Freud and Rie said that there is hardly an infectious disease that may not at times cause acute encephalitis with paralysis. They said further that vaccination against smallpox has in several instances produced infantile cerebral palsy, and cited cases reported by von Heine, Osler, Winllamier, Sachs and Peterson and Marie. Osler described several cases of encephalitis in reports on typhoid fever from the Johns Hopkins Hospital. Dysentery and pneumonia, mumps and purpura have been followed by encephalitis. Vaccinal encephalitis, which occurs more frequently at the present time, was considered in a paper by Freud and Rie on cerebral palsies.

Leichtenstern (1885 and 1886) described nonsuppurative encephalitis in association with epidemic cerebrospinal meningitis.

DR. T. C. HEMPELMANN: As always, Dr. Abt is quite right in his reference to the length of time such cases have been noted; references in the literature go back probably more than 100 years to cases that, so far as we can judge, are similar to the ones we see now.

Dr. Abt spoke of typhoid fever. We had a large group of cases in which I, at least, felt sure we were dealing with a type of encephalitis following typhoid and which were similar to those that I have discussed. I was somewhat reluctant to include these cases in the present study, because I realized that they might open up a field for discussion, since some of them progressed from a delirium during the febrile period of typhoid to a posttyphoid state of dementia or psychosis, or, as I thought, to definite encephalitic symptoms. It was difficult to say just when the latter first occurred. Typhoid fever is so common in our hospital in St. Louis that we had thirty-five patients hospitalized at the same time; when I asked the nurse how many of those who had normal temperatures were abnormal mentally, she threw up her hands and said, "Sometimes I think they are all goofy." As we went through the wards, I had to admit that many of them looked "goofy." One child in particular looked like an idiot. I showed him to a postgraduate group, and I said that he had probably always been mentally deficient. We did not have a history. Before the end of the month, I showed the same child, perfectly normal mentally. I think that this case belongs in the group that I discussed.

Dr. Neff mentioned the frequency with which these cases are diagnosed as meningitis. Neurologists almost invariably call the condition meningitis, particularly tuberculous meningitis, if they know that there are a few mononuclear cells in the spinal fluid. It is sometimes difficult to make them change the diagnosis. I am inclined to think that the case Dr. Neff mentioned, which followed unsuccessful vaccination, fits in with the one we saw coincident with pyelitis, which had nothing to do with the development of encephalitis. Some mild infections of the upper respiratory tract are easily overlooked.

Dr. Brennemann spoke of the increased frequency of encephalitis, which corresponds with the experience of almost every one who has reported cases of postinfectious encephalitis. It seems to appear in waves. When one sees two or three cases, one is apt to find more in the near future, or to know of other investigators who are seeing cases at about the same time.

Sixth Session

Afternoon, April 17

THE PROGNOSIS OF ACUTE ENDOCARDITIS IN CHILDHOOD. DR. JOHN LOVETT MORSE, BOSTON.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

DISCUSSION

Dr. M. McCulloch, St. Louis: It is a pleasure to hear a paper like this from Dr. Morse, because few of us have had the opportunity to see our patients long enough to follow them through more than one decade. Dr. Holt was one of the first to point out that children can outgrow heart disease. This question is much debated among cardiologists, and to have Dr. Morse bring together such a large group of children who have apparently outgrown, to all intents and purposes, their original lesions is a distinct contribution to our knowledge on this subject.

I think that in studying such a group of cases it makes a difference whether one includes only those who have the more serious forms of heart disease or all cases as they appear in a period of time, as Dr. Morse indicated. If all the cases are included as they appear, many patients will be seen when they have a first attack of rheumatic fever with endocarditis. In such children, other attacks may not occur, and they will ultimately recover. If, however, only children who show structural organic heart disease with endocarditis or those who have subsequent attacks of rheumatic fever are included, the prognosis is more serious. For example, in our group of cases the children who have had mitral stenosis have uniformly done poorly; after a period of five or more years, most of them are dead. In a recent report by Cotton it was shown that about 65 per cent of the children who had mitral stenosis as long ago as ten years were dead at the present time, whereas only about 27 per cent of a group of children who had mitral insufficiency alone were dead. It seemed to us that if only the children who are seen during the first attack, with a mitral murmur without much cardiac enlargement, are included, a large number will be found to get well. If you wait until they have further attacks of rheumatism, the prognosis becomes increasingly worse, because there is a tendency toward the development of more serious lesions in the heart.

I was interested in Dr. Morse's remarks concerning pericarditis. We have observed that, at the end of about five years, certain patients with pericarditis are apparently well, particularly those with acute serofibrinous pericarditis. Those who have more extensive changes in the pericardium with proliferative pericarditis do even more poorly than those who have mitral stenosis.

At the meeting of the American Heart Association in Detroit, last year, Dr. Lewis Conner pointed out the fact that a group of patients with aortic insufficiency without mitral stenosis due to rheumatism, who had lived many years into adult life, seemed well and had a good prognosis. We ordinarily think of aortic insufficiency as being due to syphilis. In most adults aortic insufficiency is caused by syphilis, but children with rheumatic heart diseases frequently have aortic insufficiency with or without mitral stenosis. In our cardiac clinic we have been impressed with the observation Dr. Conner pointed out, and we have observed a number of patients who still have signs of aortic insufficiency and are doing extremely well after ten years. The tendency for children to grow and to replace scar tissue with normal tissue is a factor of considerable importance that is often overlooked in discussing the prognosis of heart disease in children.

Dr. H. F. Helmholz, Rochester, Minn.: I was glad to hear Dr. Morse emphasize the matter of rest in the treatment for acute endocarditis. I well remember a paper and its discussion at the Washington meeting of the American Medical Association, in which the author reduced the period of rest to a few weeks. I think that the paper presented is a vindication of the stand Dr. Morse took at the time. I think that in rest we have probably the most important element of success in the treatment for acute endocarditis.

Dr. I. A. Abt, Chicago: I wish to refer to the cases of two children with aortic insufficiency whom I took care of years ago; both of them are still alive. One, now a man in his early thirties, was taken ill at about the age of 11 years with rheumatism, which manifested itself as abdominal pain. Aortic insufficiency developed. He now has a competent heart and is actively engaged as a lawyer.

The other case was that of a girl whom I saw about her eleventh or twelfth year, in whom aortic insufficiency developed. She must be close to 35 now; she has borne several children, and she is still well.

These two patients have not shown ill effects from the rheumatic lesion of the heart acquired in childhood.

DR. J. L. MORSE: I want to emphasize what I mean by rest. The children who had perfect care stayed in bed for a period of three months as a minimum. Many of them were in bed for six months. They were carried upstairs for six months or a year afterward, and were allowed to walk only on a level surface. They were kept out of sports and athletics for many years, and then were gradually allowed to participate in them.

For example, one patient, who is now an officer in the Navy and is perfectly well, said he remembered walking into my office when he was a little boy, of being taken off the office table in his father's arms and of not setting his feet on the ground again for six months. That is what I mean by care.

As an example of what may happen in the worst cases, I shall detail one case: A boy from a poor family, whom I saw in consultation, had endocarditis, myocarditis, pericarditis, an effusion in the pericardium, pleurisy on the left side and a compressed lung. I finally located him, and he came to see me, about twenty years after we had examined him. He said that he had never had any special care; from the beginning he got up and did as he pleased, or as much as he felt able to do. His father was a coal dealer, and, after he was 13 years old, he worked after school in the coal yard. While he was in high school, he played on the baseball team, was captain of the football team and played on the basket ball team. After he finished school, he got married; he then went into the coal business for himself and has been in it ever since. For amusement, he plays semiprofessional football in the summer and boxes in the winter, sometimes for stakes. He is the most magnificent specimen of manhood I have ever seen, with an absolutely normal heart. I had Dr. Wyman, Dr. Hill and Dr. Baty listen to it, and they all agreed that it was normal in size and had perfectly good sounds.

HABIT SPASM: A PHYSICAL MANIFESTATION OF EMOTIONAL REACTION RESULTING FROM ENVIRONMENTAL STIMULI. DR. BORDEN S. VEEDER, St. Louis.

Habit spasm is a condition that is characterized by physical manifestations. Its origin, however, is in an emotional reaction resulting from some stimulus in the child's environment to which the child fails to adjust. The emotional conflict finds an outlet or expression through the muscular system. A change in environment leading to an elimination of the stimulus removes the emotional conflict, and the habit spasm disappears. Fear, inferiority, religious emotion, etc., may cause the emotional conflict and, in turn, the habit spasm. The study and approach to the condition from this point of view emphasize the necessity for the pediatrician to have an interest in and a knowledge of the abnormal psychology of childhood.

CHANGES IN THE WATER CONTENT OF THE BLOOD DURING THE COURSE OF EDEMA OF RENAL TYPE IN SEVEN CHILDREN. DR. WILLIAM B. MCCLURE, CAROL BEELER DE TAKATS (by invitation) and WINIFRED FRANZ HINMAN (by invitation), Evanston, Ill.

In six cases, the relative volume of plasma, which was determined during the development of edema, was lower than the average normal in at least one determination in each. When tested again in four patients a few days before the edema began to subside, it had increased in three and decreased in one. When examined in five patients during or just at the end of subsidence, it had increased as compared with the developmental stage (in two it exceeded the average normal).

Plasma solids, which were determined in five cases, were always low when definite edema was present. The nonprotein plasma solids were usually above normal.

Cell solids, which were determined in four cases, varied at times in the same patient, usually in a direction opposite to the changes in the relative cell volume, suggesting a movement of water in and out of the cells.

At times, the hemoglobin varied inversely to the relative cell volume, possibly due to swelling or shrinking of the cells from a change in their water content.

When the edema was increasing the whole blood solids were above the average normal in four cases and below in two, and they were lower when the edema was subsiding. In general, they were highest when the relative volume of plasma was lowest, and they decreased when the volume increased.

Moore and Van Slyke found from 5.2 to 5.8 per cent of total protein and from 2.3 to 2.7 per cent of albumin, the critical level below which edema was usually present. In our cases the percentages of total protein and albumin in the plasma (when determined) were always low when definite edema was present. The percentage of total protein in the plasma, which was tested in four cases during the development of edema was below the critical level. In three cases reexamined shortly before loss of edema began, the percentages were increased in one, decreased in one and unchanged in one, the highest being 5.15. During loss or at the end of edema, the percentages in four cases did not exceed 4.8, and in two they were 5.5 and 6.45, respectively. Moreover, in a series of tests during another attack of edema, the latter case showed a decrease instead of an increase of protein when the edema was subsiding. The albumin in the plasma, which was estimated in four cases, did not exceed 1.95 per cent during any stage of the edema, including three tests in two cases during the subsidence or at the end of the edema. These findings, during improvement, cast doubt on the importance of low blood plasma proteins as the primary or active cause of edema. Certainly some other factor (or factors) was active in our cases in inaugurating the loss of edema. However, it is probable that low osmotic pressure from low plasma protein favors the working of these factors in producing edema.

Our findings, in general, seem to indicate that with the development of edema, a loss of blood plasma occurred. The remaining plasma was diluted. This reduction in the volume of plasma is probably due mostly to a retention of water in the general tissues. Just preceding the loss and during the loss of edema, an increased relative volume of plasma occurred, probably due mainly to a movement of water from the tissues to the blood. The movement of water from the blood cells to the plasma and the reverse are possible factors in the changes of the relative volume of plasma.

THE FAT OF BREAST MILK: ITS COMPOSITION, ORIGIN AND IMITATIONS. DR. L. E. HOLT, JR., DR. H. C. TIDWELL (by invitation) and DR. W. M. COX, JR. (by invitation), Baltimore.

The belief that breast milk fat is more readily digested by infants than cow's milk fat rests on: (1) clinical impressions and (2) a certain amount of chemical evidence, but (3) chiefly on known physical and chemical differences between the two fats. The emulsified particles of breast milk fat are said to be finer and more uniform; breast milk fat contains more of the unsaturated oleic acid and less of the volatile short-chain fatty acids. Imitations of breast milk fat in which attempts have been made to overcome this difference have been widely used: (1) homogenization, to reduce the size of the emulsified particles, (2) heating, to remove the volatile acids, as is done in the various butter-flour mixtures, and (3) the use of various mixtures of animal and vegetable fats by which the percentage of unsaturated fat is increased and that of the short-chain fats is diminished.

Our observations indicate that the physical and chemical differences between the two fats are not as constant as has been believed. From microscopic examinations of single specimens it is not possible to distinguish breast milk from cow's milk. A chemical examination of milk fat from forty American women showed striking differences from the accepted German values on which the imitations of the breast milk fat have been based, notably a higher iodine value. These differences are attributed to the diet of the mother.

The available evidence indicates that, unlike the other components of milk, the fat of milk is closely similar to the neutral fat of the blood serum, and that comparatively little change occurs during its secretion by the mammary gland.

The variations to which milk fat is subject would suggest that to imitate the fat of women's milk on a particular diet, as has been done hitherto, is an arbitrary procedure of questionable value.

DISCUSSION

DR. H. J. GERSTENBERGER, Cleveland: Did the mothers who made up Dr. Chapins' group receive cod liver oil? We have found high values for iodine, I believe as high as 59, in the fat of the milk of some of our wet nurses who received cod liver oil or viosterol. I am sorry that I forgot to bring with me notes containing definite information regarding our isolated experiences on this point during the past few years. I am especially chagrined, because I brought this material with me for use in this discussion. Under the circumstances, I cannot be absolutely certain regarding the exact level of the values for iodine found by us in these cases, but I believe that I am correct in the statement that they were decidedly above the 46 to 48 reported by Arnold in the literature and used by us as our guide.

I do not think that Dr. Holt's failure to differentiate between S. M. A., Recolac and Similac is quite fair. S. M. A. was an attempt on my part to modify the fat in a manner to make it more like the fat of human milk than is the fat of cow's milk, whereas Recolac and Similac are commercial imitations of S. M. A., the fats of which give character numbers entirely different from those of the fat of S. M. A. or of the fat of human milk. The table that Dr. Holt showed on the screen clearly demonstrated this difference, but he did not call attention to it.

I do not think that Dr. Holt's statement that he does not think it worth while to attempt to imitate human milk, because such a food has not been scientifically proved to be of superior value, is necessarily correct.

Even though at times one may not be able to present scientific proof of the value of a method or of a substance, nevertheless, when a practical test on an extensive scale has without question demonstrated its success, one may be justified in accepting that its value is real. Not so long ago, when both fat and lactose were given in liberal amounts, they were frequently considered poisonous to infants, and yet we have been able to put a high fat content and a high lactose content in a food mixture low in protein, and to see it succeed as no other similar modification did before it. It seems to me that this extensive practical experience with S. M. A., over what is now a ten year period of general use, indicates that there may be something good and desirable in adapting an artificial food mixture to human milk.

DR. L. E. HOLT, JR.: In regard to Dr. Gerstenberger's question, the mothers were not receiving appreciable quantities of cod liver oil. Some were of Italian extraction and had been taking liberal amounts of olive oil. Others ate considerable amounts of carbohydrate.

I did not intend to compare S. M. A. with other fat mixtures, or to criticize the skill with which in this preparation certain of the fat constants were obtained with values very close to the figures given in the literature for breast milk. My point was that there is nothing sacred about the accepted figures for the fat constants of breast milk. I am not convinced that the values given are by any means essential or that they represent an optimum for the nutrition of the child.

There is reason to believe that it may be desirable to give a milk with a higher content of iodine than that of breast milk. The infant is born with a body fat much more saturated than that of the adult. At birth, the depot for fat has an iodine content of about 45; during the first year it rises, and at the end of this period it approaches the adult value of from 65 to 70.

True sclerema neonatorum, a condition of hardening of the fat in the body, is seen only during the period when the iodine content of the subcutaneous fat is low. The character of the fat in the body is undoubtedly not the only factor in the pathogenesis of this disease, but it is one factor. Perhaps, if infants were fed olive oil or a fat with a high iodine content from the beginning, there might be even fewer cases of sclerema.

DR. GERSTENBERGER: S. M. A. aims to raise the iodine content, not to lower it.

A Probable Deficiency Disease of Beriberi Type. Dr. Warren S. Sisson.

This paper was read by title.

The Cure of Infantile Rickets with Tungsten-Filament Radiation. Dr. Henry J. Gerstenberger and Dr. Arthur J. Horesh (by invitation), Cleveland.

As the results of investigations made by us in the past have convinced us that actinic rays and human skin, and not diet and the gastro-intestinal tract, are nature's combination for preventing rickets in the human infant, as ordinary glass bulb tungsten-filament lamps of 500 watt intensity are being used today in the practice of illumination, and as it would be ideal to receive during the process of practical illumination the benefit of the antirachitic wavelengths, we were glad to avail ourselves of the opportunity offered by M. Luckiesh, director of the Lighting Research Laboratory, Nela Park, Cleveland, to study the antirachitic value of the CX 500 watt Mazda lamp on rachitic human infants. Dr. Luckiesh has coined the term "dual-purpose lighting" for this kind of illumination, i. e., lighting for vision and for health.

In addition to determining the antirachitic value of this lamp for human infants, we were interested in establishing, if possible, a method of exposure that would be feasible from the standpoint of practicability and from that of harmlessness. At the same time, we also had in mind the desirability and possibility of making, from successful results obtained from the exposure of the limited areas of skin, as chosen for the infants, practical deductions for use in the preventive illumination of children and adults, for whom, because of the failure of rickets to develop, it has as yet been impossible to obtain scientific evidence for the need of exposure to the actinic rays.

The results were as follows:

1. Three infants with moderately severe rickets (two colored and one white) were cured (as determined by examination of the blood and x-rays) by daily twelve hour exposures to suberythemal doses of tungsten-filament radiation produced by a 500 watt CX Mazda lamp suspended in oxidized aluminum reflectors from the ceiling of a room at a distance of 5½ feet (167.6 cm.) about the level of the bed, representing a total daily illuminating intensity of approximately 900 footcandle-hours, or an average of intensity of illumination of 75 footcandles.

2. The infants were dressed in a shirt, diaper and booties, the head, neck and greater part of the extremities being exposed. Goggles were used during the first few days, but were discarded without harm because of the annoyance produced by th moisture that collected beneath them.

3. Erythema was never observed, though a mild but definite pigmentation gradually developed over the exposed areas.

4. The colored infants perspired rather regularly, whereas the white infant usually showed sweating only after taking the bottle. This difference was undoubtedly due to the increase in absorption and the decrease in reflection of the rays by the skin of the colored infants, whose bodies responded as physical black bodies.

5. No discomfort was noticed in the eyes, on the skin or in the mucous membranes. Infections of the upper respiratory tract that were present when the observations were begun were not prevented from recurring.

6. Further observations are being made with exposures to a smaller number of footcandle-hours.

7. These observations, especially in view of the fact that the curative dosage is of necessity larger than the preventive dosage, indicate that the "dual-purpose lighting" as proposed by Luckiesh probably will become feasible practically, especially for children and adults who spend the days of fall and winter within buildings.

8. The front and back of a fourth infant was exposed at a distance of 80 cm. for one and one-half hours to an intensity of illumination of 1,200 footcandle-hours produced by a portable unit, with resultant complete healing (as determined by studies of the blood and x-rays) of the moderately severe rickets within seven and one-half weeks.

DISCUSSION

DR. B. S. VEEDER, St. Louis: I did not understand whether the lamp is a special one that is being made for irradiation, or whether it is one of the commercial lamps put on the market for illumination.

DR. H. J. GERSTENBERGER: I believe that this lamp is on the market, and I think that any one can buy it. It is an ordinary 500 watt tungsten-filament Mazda lamp, housed in a glass, which permits the passage of ultraviolet rays to a wavelength of 2,800 angstrom units. It is frosted on the inside. The life is supposed to be 500 hours. Lighting engineers maintain that they can improve on nature in lighting a house, and that it is cheaper to illuminate a house satisfactorily by artificial means of illumination. Mr. Luckiesh states that we need windows today only to give psychic stimulation and rest and not to give light for vision. He maintains that if we would get along without windows, the building of homes and buildings would be cheaper, and that we can get better illumination and ventilation without windows than with them.

Book Reviews

L'INVAGINATION INTESTINALE AIGUË DU NOURRISSON. By F. D'ALLAINES, Chirugien des Hôpitaux de Paris, Agrégé à la Faculté, and JEAN ARMINGEAT, Acien Interne, Lauréat des Hôpitaux de Paris (Médaille d'Or), Chef de Clinique Adjoint à la Faculté. Paper. Price, 22 francs. Pp. 122, with 12 illustrations. Paris: Gauthier-Villars & Cie, 1931.

This excellent little monograph (one of a series of pediatrics topics) covers the subject of acute intestinal intussusception thoroughly, completely and clearly. It will prove of equal interest and value to the pediatrician, surgeon and general practitioner with a reading knowledge of French. Following a commendatory brief preface by Professor Ombrédanne, the authors in concise and orderly manner take up in ten chapters pathologic anatomy and physiology, etiology, symptoms, progress, clinical types, prognosis, diagnosis and treatment. A special chapter is devoted to surgical treatment and the variations in surgical procedures, with variations in the condition present, especially as regards the length of time the condition has existed. The final chapter is devoted to a brief exposition of the mortality statistics and the end-results of the varying types of operative procedures over a period of years. There is a moderate bibliography given in footnotes which might better have been collected and placed at the end. The roentgen diagnosis of the condition is well stressed and illustrated with typical examples. The various surgical steps and procedures are briefly outlined and perhaps could have been bettered with a few more diagrams. Quite naturally, the tenor of this entire essay is an insistent plea for early recognition and treatment of the condition. In the facts and procedures assembled, there is nothing especially new and no radical departure from those in use in all the better clinics. However, as a brief and logical review of this ever pertinent subject, this monograph is highly recommended.

THE INFANT WELFARE MOVEMENT IN THE EIGHTEENTH CENTURY. By ERNEST CAULFIELD. Price, $2. Pp. 203, with 8 illustrations. New York: Paul B. Hoeber, Inc., 1931.

The author gives the story of the progress made in infant welfare in London during the eighteenth century. He gives in detail the reasons for the tremendous infant mortality at the beginning of the century, and develops this to the opening of the Foundling Hospital by Thomas Coram. He gives this movement in detail with the work of William Cadogan, Jonas Hanway and George Armstrong.

Much of the literature of the time has been consulted, and there is a complete bibliography. This book fills a place in the history of the development of infant welfare. Strangely enough the beginning steps made in the eighteenth century have not been fully enough realized, and this justifies the writing of such a book. It is well written and bound attractively, and should have a place in the library of any one interested in the development of pediatrics.

Directory of Pediatric Societies

INTERNATIONAL

International Association of Preventive Pediatrics

Secretary: Prof. G. Scheltema, 14a Peolstraat, Groningen, Holland.
Place: The Hague. Time: September 7-8, 1931.

International Pediatric Congress

President: Dr. George Frederic Still, 28 Queen Anne St., London, W. 1.
Place: London. Time: 1933.

NATIONAL

American Medical Association, Scientific Assembly, Section on Diseases of Children

Chairman: Dr. Jay I. Durand, 1305, 4th Ave., Seattle.
Secretary: Dr. Alfred A. Walker, Highland Plaza Apartments, Birmingham, Ala.
Place: New Orleans. Time: 1932.

American Academy of Pediatrics

President: Dr. John L. Morse, 319 Longwood Ave., Boston.
Secretary: Dr. C. G. Grulee, 636 Church St., Evanston, Ill.
Place: New Orleans. Time: June, 1932.

American Child Health Association

President: Hon. Herbert Hoover, Washington, D. C.
Secretary: Dr. Philip Van Ingen, 125 E. 71st St., New York.

American Pediatric Society

President: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Secretary-Treasurer: Dr. Hugh McCulloch, 500 S. Kingshighway, St. Louis.
Place: Rochester, Minn. Time: May 26-28, 1932.

Association of American Teachers of the Diseases of Children

President: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.
Secretary: Dr. Oscar Reiss, 1930 Wilshire Blvd., Los Angeles.
Place: New Orleans. Time: 1932.

Children's Hospital Association of America

President: Dr. Joseph Brennemann, 707 Fullerton Ave., Chicago.
Secretary-Treasurer: Miss Bena M. Henderson, Milwaukee Children's Hospital, Milwaukee.
Place: Toronto. Time: September, 1931.

Canadian Society for the Study of Diseases of Children

President: Dr. G. A. Campbell, 164 Metcalfe St., Ottawa, Ontario.
Secretary-Treasurer: Dr. L. P. MacHaffie, 162 Clemow Ave., Ottawa, Ontario.

SECTIONAL

Central States Pediatric Society

President: Dr. A. Graeme Mitchell, 345 McAlpine Ave., Cincinnati.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Blvd., Chicago.
Place: Cincinnati. Time: Oct. 16-17, 1931.

Southern Medical Association, Section on Pediatrics

Chairman: Dr. D. Lesesne Smith, 188 S. Church St., Spartanburg, S. C.
Secretary: Dr. Ludo von Meysenbug, 3439 Prytania St., New Orleans, La.
Place: New Orleans. Time: November, 1931.

STATE

Alabama Pediatric Society

President: Dr. Thomas J. McElhenney, 1722, 2nd Ave., Bessemer.
Secretary-Treasurer: Dr. John W. Simpson, 1117 S. 22nd St., Birmingham.
Place: Mobile. Time: April 20, 1932.

Georgia Pediatric Society

President: Dr. Charles E. Boynton, 118 Forrest Ave., N. E., Atlanta.
Secretary-Treasurer: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N. E., Atlanta.

Louisiana State Pediatric Society

President: Dr. R. T. Lucas, Shreveport, La.
Secretary-Treasurer: Dr. Suzanne Schaeffer, Chailie Bldg., New Orleans.

Oklahoma State Pediatric Society

President: Dr. C. E. Bradley, Medical Arts Bldg., Tulsa.
Secretary: Dr. G. H. Harrison, 1200 N. Walker St., Oklahoma City.
Place: Tulsa. Time: May 14, 1932.

South Carolina Pediatric Society

President: Dr. E. W. Barron, 1512 Marion St., Columbia.
Secretary-Treasurer: Dr. Julian P. Price, 117 W. Cheves St., Florence.

Texas Pediatric Society

President: Dr. J. H. Park, Jr., 3717 Main St., Houston.
Secretary-Treasurer: Dr. P. E. Luecke, 4105 Live Oak St., Dallas.

Virginia Pediatric Society

President: Dr. W. B. McIlwaine, Petersburg.
Secretary: Dr. J. B. Stone, 2042 Park Ave., Richmond.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. O. L. Goehle, 15701 Detroit Ave., Lakewood, Ohio.
Secretary: Dr. W. C. Fargo, 8314 Euclid Ave., Cleveland.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. Gladys Boyd, 56 DeLisle St., Toronto.
Secretary: Dr. Lawrence Murray, 1870 Bloor St., W., Toronto.

Bronx Pediatric Society

President: Dr. Joseph Golomb, 496 E. 175th St., New York.
Secretary: Dr. E. Leonard Benjamin, 2229 Valentine Ave., New York.
Place: Concourse Plaza, 161st St. and Grand Concourse. Time: Second Wednesday of each month, except June, July, August and September.

Brooklyn Pediatric Society

President: Dr. Benjamin Kramer, 6 Pierrepont St., Brooklyn.
Secretary: Dr. Lambert Krahulik, 172 Clinton St., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Fourth Thursday of October, January and April.

CHICAGO PEDIATRIC SOCIETY

President: Dr. Henry E. Irish, 432 S. Lincoln St., Chicago.
Secretary: Dr. Bert I. Beverly, 8 S. Michigan Ave., Chicago.
Place: City Club. Time: Third Tuesday of each month.

DALLAS COUNTY PEDIATRIC SOCIETY

President: Dr. R. S. Usry, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. P. E. Luecke, 4105 Live Oak St., Dallas, Texas.
Place: Bradford Memorial Hospital. Time: 1 p. m., first and third Saturdays of each month.

DETROIT PEDIATRIC SOCIETY

President: Dr. Allan L. Richardson, 651 Fisher Bldg., Detroit.
Secretary: Dr. Joseph A. Johnston, Henry Ford Hospital, Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

FULTON COUNTY MEDICAL SOCIETY, PEDIATRIC SECTION (ATLANTA, GA.)

Chairman: Dr. James A. Wood, Medical Arts Bldg., Atlanta.
Secretary: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N.E., Atlanta.
Place: Academy of Medicine, 32 Howard St. Time: Second Thursday of each month from October to April, 8 p. m.

MEMPHIS PEDIATRIC SOCIETY

President: Dr. Kinsey M. Buck, 1024 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. R. B. McCormick, 1074 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

MILWAUKEE PEDIATRIC SOCIETY

President: Dr. H. O. McMahon, 84 E. Wisconsin Ave., Milwaukee.
Secretary: Dr. F. R. Janney, 2018 E. North Ave., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

NEBRASKA PEDIATRIC SOCIETY

President: Dr. H. B. Hamilton, 434 Aquila Court Bldg., Omaha.
Secretary: Dr. George L. Clark, 1817 Vinton St., Omaha.
Place: As announced by committee. Time: Second Thursday of each month from October to June, inclusive, 6 p. m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. Elmer W. Barron, 520 Commonwealth Ave., Boston.
Secretary: Dr. Gerald Hoeffel, 319 Longwood Ave., Boston.
Place: Boston Medical Library. Time: October, January and March.

NEW YORK ACADEMY OF MEDICINE, SECTION OF PEDIATRICS

Chairman: Dr. Herbert B. Wilcox, 39 E. 75th St., New York.
Secretary: Dr. John P. Caffey, 310 Riverside Drive, New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. Eber D. Kanaga, Townsend Bldg., Tacoma, Wash.
Secretary: Dr. Homer T. Clay, Washington Bldg., Tacoma, Wash.

Northern California Pediatric Society

President: Dr. Edward B. Shaw, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. Lloyd Hardgrave, 384 Post St., San Francisco.
Place: Various teaching schools. Time: Monthly.

Northwestern Pediatric Society

President: Dr. E. S. Platou, Medical Arts Bldg., Minneapolis.
Secretary: Dr. F. G. Hedenstrom, 625 Lowry Bldg., St. Paul.

Philadelphia Pediatric Society

President: Dr. John P. Scott, 2040 Pine St., Philadelphia.
Secretary: Dr. John D. Donnelly, 115 Bryn Mawr Ave., Bala, Pa.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

Pittsburgh Pediatric Society

President: Dr. David H. Boyd, 200, 9th St., Pittsburgh.
Secretary: Dr. Theodore O. Elterich, 724 Highland Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate months from October to April, inclusive.

Richmond Pediatric Society

President: Dr. Henry S. Stern, 807 W. Franklin St., Richmond, Va.
Secretary-Treasurer: Dr. W. C. Chapin, 922 Floyd Ave., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St. Time: First Thursday of each month (other than during the summer).

Rochester Pediatric Society

President: Dr. S. W. Clausen, Strong Memorial Hospital, Rochester, N. Y.
Secretary: Dr. F. W. Bush, 221 S. Goodman St., Rochester, N. Y.
Place: Arranged by program committee. Time: First Friday of each month from October to May.

Rocky Mountain Pediatric Society

President: Dr. Wilford W. Barber, 227, 16th St., Denver.
Secretary: Dr. John A. Schoonover, 1616 Tremont Pl., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month from September to May, inclusive, 2:30 p. m.

St. Louis Pediatric Society

President: Dr. Gustave Lippmann, 3720 Washington Blvd., St. Louis.
Secretary-Treasurer: Dr. Katherine Bain, 3720 Washington Blvd., St. Louis.
Place: St. Louis Children's Hospital. Time: At least once a year and contingent thereafter on the arrangement of a suitable program.

Seattle Pediatric Society

President: Dr. Herbert E. Coe, 509 Olive St., Seattle.
Secretary: Dr. Raymond H. Somers, 1305, 4th Ave., Seattle.
Place: College Club. Time: Third Friday of each month, 7 p. m.

Southwestern Pediatric Society

President: Dr. William M. Happ, 523 W. 6th St., Los Angeles.
Secretary: Dr. Jeanette Harrison, 1136 W. 6th St., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. E. P. Russell, Berwyn, Ill.
Secretary: Dr. John P. Parsons, 320 S. Division St., Ann Arbor, Mich.